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POSTANESTHESIA CARE UNIT PROBLEMS

Gerald Shapiro, MD, MPA, *Guest Editor*

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The anesthesiologist's practice has been immeasurably eased by development of the recovery room. Postanesthesia issues involving the return of sensorium, pain, and fluid-electrolyte balance must be addressed. Often, problems originating in the operating room become manifest (blood loss, neuromuscular blockade, and so forth) and must be treated. Here, also, we see specific medicolegal risk management issues. Although it is not the objective of this presentation to review "the law" exhaustively, some of the most pertinent administrative and legal situations facing the recovery room director are presented.

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Preface

The central theme for this issue is different than any other. This is not a do-it-yourself book. The focus is on the management of postanesthesia problems. In its fundamental sense, to understand problems is to know how to prevent harm and thus protect the patient. No sensible choice of patient management can be made without careful analysis of the advantages and disadvantages of each course of action. How to conduct such an analysis is the subject of this issue.

This treatise is dedicated to all those who continue to strive for the consideration that postanesthesia care has a high priority in the practice of medicine. Without these individuals and organizations (physicians, postanesthesia care nurses, CRNAs, American Society of Anesthesiologists, JCAHO, state agencies, and others), recovery room patient management would have continued its lackluster existence. Because of great strides, predominantly in monitoring technology and knowledgeable observation, fewer complications befall the postanesthetic patients, more complex surgery can be performed, and there is an earlier and more responsive reaction to problem solving.

This issue brings together the finest authorities. Working with them has been a pleasure. I hope they forgive me for hounding them.

There are always behind-the-scenes people to thank, those who make everything come together—Jacob Israel, MD, advisor and friend who helped select the authors; Margaret Burns of W. B. Saunders who did not scream when the deadline passed, but rather encouraged my endeavors; to my Carole who gave her time to provide her special rhetorical touch; and to my mother and dad, both deceased, who encouraged me in my early career to focus on postanesthesia care because so few were.

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FORTHCOMING ISSUES

September 1990

HEMORRHAGIC DISORDERS
Bruce Spiess, MD, *Guest Editor*

Anesthesiologists administer an enormous amount of blood products. Although this transfusion practice has well-known major, immediate, and late implications and complications, the recent AIDS crisis has put transfusion practices under intense review and rapid change where appropriate. For example, there are now new NIH blood product utilization guidelines, new operating room coagulation function monitors, and much greater understanding of blood physiology. Thus, a contemporary, in-depth examination of transfusion and hemorrhage therapy in the operating rooms of the 1990s is a timely and essential contribution to the anesthesia literature.

December 1990

PREANESTHETIC ASSESSMENT
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March 1991

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December 1989

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Arnold J. Berry, MD, *Guest Editor*

September 1989

ANESTHESIA AND NEW SURGICAL PROCEDURES
A. Scott Wheeler, MD, *Guest Editor*

Foreword

All anesthesiologists agree that the induction of and emergence from anesthesia are the most dynamic, complication-laden, disaster-prone times during the perioperative period for most patients. Further, if a given patient comes to anesthesia and surgery already critically ill, and it is hoped that they can be returned to a high-quality meaningful life immediately postoperatively, then the management of the induction of and emergence from anesthesia is even more perilous and critical. To a very significant extent the emergence from anesthesia is managed in the postanesthesia care unit (PACU) and therefore, the proper management of patients in the PACU is one of the most important and pressing concerns of anesthesiologists.

How should a PACU be administered? It seems clear that today's PACU must and should be similar to any intensive care unit in terms of staffing, staffing credentials, vigilence, and technology. What special hemodynamic, ventilatory, fluid and electrolyte, temperature, and neurologic problems do these patients face? Certainly the very young and old may respond to this dynamic period differently than young and middle-aged adults. The management of acute postoperative pain in the patient emerging from anesthesia is of paramount importance per se, but it is also important because the proper treatment of PACU pain may greatly diminish some of the problems mentioned previously. Finally, obstetric and ambulatory surgery patients have their own special PACU problems that arise from their special surgical environments. Since the PACU is a complication-prone place, it should come as no great surprise that the PACU is the birthplace of many medicolegal problems. This issue of *Anesthesiology Clinics of North America* on PACU Problems completely and in great depth discusses all of these issues.

Dr. Gerald Shapiro is eminently qualified to guest edit this important issue of *Anesthesiology Clinics of North America.* His special interest in PACU problems began when as a cardiac anesthesiologist he was solely responsible for the postoperative care of cardiac surgery patients. As a result of this intense experience with PACU problems, he soon became the founding father (by ASA appointment) of the American Society of Post-Anesthesia Nurses Association, a co-author of the JCAH Accreditation Manual for hospitals, a member of the ASA Operating Room Committee and the Allied Health Care Committee, directed numerous national ASA recovery room workshops, and presently is widely sought after by various institutions (hospitals and states) to consult on PACU problems. As a natural consequence of this impressive list of PACU activities, Dr. Shapiro has lectured and written extensively on PACU problems. As a further consequence of his central role in dealing with PACU problems, he knows who are the important workers in this area and has gathered a most qualified and knowledgeable list of contributors. Because of the timeliness and importance of the PACU problems subject and the expertise of Dr. Shapiro and the excellent group of contributors that he has gathered, the practice of all anesthesiologists will greatly benefit by their reading this issue of *Anesthesiology Clinics of North America.*

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Postanesthesia Care Unit Administration and Staffing

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The postanesthesia care unit (PACU) is a specialized intensive care unit, and just as the coronary care unit (CCU) or surgical intensive care unit (SICU), it requires physician and nursing staff with special expertise to care for patients in their immediate postoperative phase, still under the effects of anesthesia. This article will discuss the issues of administration and staffing of the unit.

PERSONNEL AND THEIR ROLES

The Medical Director

Primary medical responsibility for the unit should reside with the anesthesiology department, which may designate one of its members as the medical director or physician coordinator for the unit. Together with the nurse manager, the medical director should set criteria for admission and discharge, and protocols for patient care and quality assurance. As practice patterns vary in the many different types of hospitals, each department should identify the needs of its hospital and set standards to meet those needs as well as those of the accrediting agencies and the American Society of Anesthesiologists (ASA) and the American Society of Post-Anesthesia Nurses (ASPAN). Communication with the surgeons for major problems or those surgically related is essential, and in those units that serve as intermediary intensive care units, the issues of when the transfer of care from one service to another occurs and who is responsible for the patient must be resolved. In teaching hospitals, the medical director or a staff anesthesiologist should supervise the care rendered by anesthesiology residents who may rotate through the PACU. If certified registered nurse anesthetists are assigned to the unit, the medical director should define their responsibilities. The monitoring in the PACU is fairly extensive and the medical director should see to it that the bioengineering staff provide the necessary maintenance and support to the PACU.

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The Nurse Manager

The nurse manager of the PACU must be a registered nurse who has had previous experience in recovery room care. PACU nursing is transient yet intense and requires constant updating of one's knowledge and skills. Thus the nurse manager must do so herself or himself, and also see that the staff remain current in their knowledge and skills. The operating room schedule may vary considerably not only from day to day but during the day, and the nurse manager must adjust staffing assignments to meet the ever changing patients' needs. Orientation programs for new staff must be developed and coordinated, and the abilities of new and current staff evaluated. The nurse manager should identify a list of competencies that the PACU nursing staff should have, and these may vary from unit to unit depending on the type of unit. For example, the list of competencies may need to be quite extensive in a large tertiary care hospital that does all types of surgery including pediatrics, but may be less extensive in an adult ambulatory unit, but should then include skills in postoperative teaching. A list of textbooks and other resources should be kept in the PACU or nursing education office. At the end of the orientation program the nurse manager should test the competencies of the new staff nurse and record those mastered and those yet to be completed in his or her file.

The nurse manager has a dual chain of command, to the nursing director and to the anesthesiology director. He or she should interact with the other nurse managers to communicate reciprocal needs and meet with the medical director on a regular basis to review care and identify problems and solutions. As the PACU has no control over the termination of surgery, the nurse manager should maintain good communication with the operating room nurse manager, to anticipate the transfer of patients, and with the receiving nursing units to move patients expeditiously from the PACU so that the operating room suite runs efficiently. This task may be delegated to a charge nurse. The nurse manager should be certified by ASPAN in postanesthesia care nursing.

The Staff Nurse

Nurses in this unit must be selected and adequately trained to render postanesthesia care. Previous experience on a surgical floor or intensive care unit is good preparation, but because of the need to recognize promptly and react quickly to the special problems in patients immediately postoperative and partially anesthetized, an orientation course of from 2 to 8 weeks is recommended depending on the individual nurse's previous experience, the volume and complexity of the cases done in that unit, and the time that can be allotted for training. The nurse needs to have a thorough understanding of local and general anesthesia as well as of the usual surgical procedures done in that hospital or surgicenter and the pathophysiology related to those procedures and the perioperative state. Familiarity with electronic monitors and mechanical ventilators is essential for those in hospital PACUs. A set of textbooks and other resources should be kept in the PACU or nursing education office. To provide clinical training, it may be useful to double team the new recruit with a clinical instructor from the nursing education department or with an experienced PACU staff nurse as a preceptor. Luczun[4] has developed a weekly modular program and cites educational guides, the responsibilities of the instructor, and an evaluation program. Although the list of competencies may be long or short according to the complexities of the surgical case load done in that particular health facility, the nurse should maintain a log of the experiences gained and the competencies as they are mastered. It is essential that all PACU nurses have current basic life support (BLS) certification, and for those who work in hospitals, it is highly recommended that they all take an advanced cardiac life support (ACLS) course, and seek ASPAN certification. It is advisable that the PACU nurse spend 1 or more days in the operating room with an anesthesiologist to obtain first-hand experience of anesthesia and surgery so as to have a better understanding of what the patients will have undergone and thus be better able to care for them. The registered nurse should be at the bedside of the patient and should not be assigned primarily clerical and housekeeping chores. Continuing education and updating of knowledge and skills are essential for the staff nurse (Table 1).

Other Staff

Licensed practical nurses, after an appropriately tailored education program with a specific list of competencies, can function well in the PACU. A licensed registered nurse should be in charge of all ancillary personnel, and the nurse manager should designate the types of patients and level of care that can be rendered by the LPN. Carefully selected and trained nursing assistants may assist in some direct patient care in activities, such as monitoring low-risk patients, under the direct supervision of a licensed registered nurse (Table 2). For those who evidence interest in postanesthesia care, it is essential that they first work in the unit and, after a period of familiarity with its function,

Table 1. *Training Program for Registered Nurses*

1. Orientation to the postanesthesia care unit
2. Airway assessment and management
3. Cardiovascular assessment, monitoring, and management
4. Arrhythmia recognition and treatment
5. Nervous system assessment, monitoring, and management
6. Fluid, electrolyte, and acid-base balance
7. General anesthetic agents, effects, and aftereffects
8. Muscle relaxant effects and assessment
9. Local anesthetic agents, techniques, and effects
10. Administration of drugs and drug-related problems
11. Monitors, ventilators, and other equipment in the PACU
12. Pediatric patients' essential differences from the adult
13. Postanesthesia nursing assessment and care plans
14. Surgery-specific nursing care plans
15. Care of the ambulatory surgery patient
16. Postoperative pain and its control
17. Miscellaneous procedures and issues in the PACU
18. Cardiorespiratory arrest and death in the PACU
19. Basic CPR and ACLS
20. Disaster protocols
21. Quality assurance and accreditation standards
22. Operating room experience
23. Policy and procedure manuals
24. Testing and evaluation of the orientation program

they should also go through an education program tailored to meet their educational needs and satisfy specific patient care competencies should it be decided that nursing assistants will be used in that capacity. Orderlies may be assigned in the PACU or may be shared with the operating room, and, if properly trained, can be used to transport well-recovered and physiologically stable patients to other nursing units. In large units, a unit secretary or clerk is valuable in handling telephones and the variety of clerical chores.

Table 2. *Training Program for Nursing Assistants*

1. The purpose of the PACU
2. Chain of command and individual responsibilities
3. Types of anesthesia, effects, and aftereffects
4. Nursing assessment and care plans
5. Monitors and other equipment
6. Routine low-risk surgical procedures and possible complications
7. Required competencies of the nursing assistant
8. Basic CPR
9. Policy and procedure manuals
10. Testing

STANDARDS OF CARE

Postanesthesia nursing care has five elements: assessment, nursing diagnosis, development of a care plan, its implementation, and evaluation of the plan. To assess a patient, the nurse obtains the essential data from the anesthesiologist or nurse anesthetist regarding the surgical procedure, anesthetic management, and all relevant medical conditions including allergies, medications, any physical or mental disabilities, and laboratory data. Additional information may also be obtained from the chart or surgeons. In the ambulatory surgery setting the nurse may be involved in preoperative interview of patients and chart review. The patient's (and family's) understanding of the procedure, the preoperative teaching, the ambulatory surgery process, and instructions for postoperative care also form part of assessment. Relevant preoperative emotional, safety, and psychosocial needs should not be omitted (Table 3).

On admission the nurse conducts a physical examination of the patient. Having completed that, the nurse makes a diagnosis based on the entire assessment, documents her findings, recording the postanesthesia score (if used), and implements a care plan in which careful monitoring and timely interventions promote the patient's safe recovery to a stable physiologic state. Attention to the other factors are given as necessary. Any alterations should be promptly recognized, noted, and communicated to the anesthesiologist or surgeon as protocol dictates. All interventions and their outcomes should be documented. If patient-controlled analgesia (PCA) is to be used, the patient should be made as comfortable as safety allows before discharge to another nursing unit. Part of PACU care may include notifying the inpatient nursing unit or family of the patient's arrival in and discharge

Table 3. *Admission Physical Examination*

1. Basic vital signs (blood pressure, pulse, respirations, and temperature)
2. Position of the patient
3. Airway patency, rate and volume of ventilation, breath sounds, and ventilator settings as indicated
4. Pressure readings as indicated (arterial, CVP, PA, PCWP, ICP)
5. Condition and color of skin
6. Condition of dressings or wound(s) if evident
7. Types and patency of catheters and drainage tubes
8. Amount and type of drainage
9. Location of lines, type and amount of fluid infusing
10. Level of consciousness (and pupillary response if indicated)
11. Muscular strength and response
12. Level of pain, physical and emotional discomfort

Abbreviations: CVP = central venous pressure; PA = pulmonary artery; PCWP = pulmonary capillary wedge pressure; ICP = intracranial pressure.

from the PACU. When the criteria for discharge are met, the nurse may send the patient out of the unit. On transferring the patient to another nursing unit, a final status report may be given to the next nurse either prior to the transfer or on arrival in that unit.

In an ambulatory surgery setting, there should be a phase 2 recovery area, and in that unit the nursing care plan includes progressive movement of the patient from a supine position, through sitting, to ambulation with assistance, and finally full ambulation. In addition to a stable physiologic state and a dry wound, the patient's ability to take fluids by mouth, have minimal or no nausea, urinate before discharge, and understand the postanesthetic and postoperative instructions should all be evaluated and clearly documented. If the patient is unable to comprehend for valid reasons, e.g., a child, then the responsible accompanying adult should be adequately instructed and it should be so documented. A follow-up telephone call the next day is highly recommended, and a postoperative mail survey can be valuable.

STAFFING

The condition of the patients and the type of surgery performed influence the need for staffing. A rule of thumb has been one nurse per two PACU beds but this guideline does not take into account varying patient needs. ASPAN has developed a patient classification that is useful (Table 4). Staffing to anticipated census and patient classification appears to be a reasonable approach to meet patient needs and fiscal responsibility. Some leeway in staffing must be allowed during high peak times for unanticipated complications, RN absences, etc., as non-PACU staff usually do not have the competencies to fill in as floats for PACU nurses. ASPAN also recommends that two nurses be present whenever there is a single patient in the unit recovering from anesthesia, and that additional staff be available when difficult patients are in the unit. PACUs open 24 hours are most likely located

Table 4. *ASPAN Patient Classification and Nursing Ratio*

Class I	One nurse to three patients who are awake and stable; uncomplicated patients
Class II	One nurse to two patients (1) Any stable unconscious patient (2) Any uncomplicated pediatric patient (3) Patients who have undergone major surgery and whose systems have stabilized
Class III	One nurse to one patient at the time of admission to the PACU for any patient requiring life support care. A second nurse must be available to assist as needed

Table 5. *Staffing According to Census for a Phase 1 Ambulatory PACU*

PATIENTS	NURSES	ANCILLARY STAFF
1	1	1
2	1	1
3	2	1
4	2	1
5	2	1
6	2	1
7	3	1
8	3	1
9	3	1

in tertiary care hospitals or may serve as back-up ICUs and may need to have their staffing patterns reviewed critically.

It is difficult to propose definitive guidelines for a hospital PACU with a mix of inpatients and outpatients because the mix may vary from day to day and from hospital to hospital, but the criteria for staffing should be to provide a nurse-patient ratio that consistently meets the needs of that unit. Operating room policies and efficiency have a significant impact on the PACU. A heavy operating room schedule on the day shift with little or no emergencies after 4 PM may require a PACU that is staffed only to 7 or 8 PM , whereas one that runs later or is inefficient with unpredictable ending times may require staffing until 11 PM or 12 AM, with the nurses on call to cover for emergencies. An option for covering emergency surgery when the PACU is closed is to hold the patients in the operating room until the anesthesiologist is satisfied that the patient is recovered sufficiently to be transferred to his or her room, or to take the patient to a nursing unit where the staff can provide the same care as in the PACU. In a tertiary care hospital or a level 1 trauma center where there is an unpredictable but fairly constant night emergency load, it is advisable that there be at least one PACU nurse and one nursing assistant in house after midnight. On those nights when the unit is empty, the nurse and assistant may be floated to an ICU. In tertiary care hospital PACUs with a preponderance of critically ill patients, it is preferable to have only registered nurses because the level of nursing care will be intense (Table 5).

ADMISSION AND DISCHARGE CRITERIA

The primary function of the PACU is to assure patients' safe recovery from the immediate effects of anesthesia and surgery, but it may serve other functions as well. The criteria for which patients go to the PACU, how they are cared for while there, and the criteria for their release from the unit should be developed by the medical director and nurse manager and clearly stated in the policy manual. Some patients, e.g., open heart cases, may bypass the PACU and go directly to the ICU, although the PACU may serve a recovery function for patients who have had angioplasty procedures rather than conventional surgery. The PACU may serve as an overflow ICU, and in some units, electroconvulsive therapy and cardioversions may be done with anesthesia. With such a potential variety of cases, it is essential that optimum care be given to each patient and staffing be consistent with their needs. Patients who are at risk for infection or who have respiratory infections or open contaminated wounds should be in an isolated recovery room or should be in a location other than the open PACU but still receive the needed level of recovery care.

Table 6. *Postanesthesia Score*

SCORE	CRITERIA	AT ADMISSION	MINUTES				AT DISCHARGE
			15	*30*	*45*	*60*	
Activity							
2	Able to move 4 extremities						
1	Able to move 2 extremities						
0	Able to move 0 extremities						
Respiration							
2	Able to breathe deeply and cough						
1	Dyspnea or limited breathing						
0	Apnea						
Circulation	Change from preanesthetic level						
2	BP ± 20%, no change in EKG or rhythm						
1	BP ± 20–50%, minor change in EKG or rhythm						
0	BP ± 50%, major change in EKG or rhythm						
Awareness							
2	Fully awake						
1	Arousable on calling						
0	Unresponsive						
Color							
2	Pink						
1	Pale, dusky, blotchy, jaundiced						
0	Cyanotic						
Total score							

Abbreviations: BP = blood pressure; EKG = electrocardiogram.
Modified from Aldrete JA, Kroulik DA: Post anesthesia recovery score. Analg Curr Rev 49:924–934, 1970.

In some units a postanesthesia score rating five standard elements is part of the initial assessment, and the anesthesia department may allow nurses to discharge patients without one of them physically present if the patient ranks 8, 9, or 10 on this score (Table 6).

On discharge from the unit, the nurse should give a report to the nurse on the receiving unit who will care for the patient and indicate the surgical procedure and any complications; the condition of the wounds, dressings and drainage sites; the type of anesthesia and any complications; the presence of nausea and vomiting; input and output; and the vital signs. If PCA was started in the PACU, the level of patient comfort and the extent of patient instructions and comprehension should be mentioned. The PACU nurse may determine which and how many persons need to accompany the patient to the next nursing unit. Critically ill patients should be transported by one or two nurses, whereas a stable fully recovered patient may be transported by an orderly only.

THE POSTANESTHESIA CARE UNIT RECORD

This record is primarily kept by the nursing staff and details all assessments, interventions, and patient responses in the PACU. Not only must notes be accurate but the exact time of events and interventions should be recorded. It is recommended that special orders, e.g., do not irrigate or move nasogastric tube, or do not resuscitate, be written on this sheet because that facilitates care of the patient. Final discharge notes by both the anesthesiologist and PACU nurse are useful, and if the postanesthesia assessment score is used, the scores on admission, discharge, and intervening times should be recorded on the chart.

PHYSICAL ASPECTS OF THE POSTANESTHESIA CARE UNIT

The open-ward concept works best for rendering care in the unit, with ceiling track curtains to provide privacy as needed. A rule of thumb is 1.5 patient stations per operating room, but this needs to be modified according to the type of surgery performed in the health care facility. This ratio works well for the average mixed-use hospital, but is inadequate for a high-volume ambulatory center where the procedures are short and recovery times may be longer. A 2 to 1 ratio may be more appropriate.

In tertiary care hospital units raw bed space should be at least 80 square feet, or allowing an 8-by-10-foot space per patient station. Separate clean and dirty utility rooms are needed, as well as sinks strategically located in the unit for hand washing. For a phase 1 ambulatory PACU, a smaller amount of space may be allocated per patient slot but not less than 54 square feet (6 by 9 feet). Although the phase 2 area may not require as large a patient slot, it is the site where family members stay with the patients and patients are encouraged to ambulate, so adequate allowance for these newer recovery area functions must be made. The phase 1 and 2 areas should be in close proximity to each other.

In a tertiary recovery area, each patient location should be equipped with at least two oxygen outlets, 3 to 4 suction outlets, and 4 to 8 110 V electrical outlets of which half should be on the emergency power supply. A shelf behind the bed or cabinet at the side of the bed for disposable supplies is useful.

At each patient console, a sphygmomanometer or automatic blood pressure monitor should be standard equipment as well as an electrocardiographic (ECG) monitor to which pressure monitoring can be added as needed. If a ventilator is not kept in the unit, one should be readily available when called for. A pulse oximeter is not yet standard in the recovery room, but these should be immediately available and used to monitor any patient who is at risk for hypoxia. Having a portable 12-lead ECG machine handy is useful. In the ambulatory setting, one oxygen and one suction machine should suffice and four electrical outlets should be ample. An ECG monitor should be at every station in the phase 1 recovery area, but a single ECG machine and one or two sphygmomanometers should suffice for the entire phase 2 area.

The nurses' station should be located so as to provide line-of-sight monitoring of every patient in the unit, if possible. It need only have drug cabinets for a limited number of parenteral medications, but federal regulations require a separate drug cabinet with double locks for narcotics and other controlled substances. A small refrigerator for medications is necessary. Appropriate space should be designated for the limited amount of clerical supplies needed, the policy and procedure manuals, and other educational resources.

The temperature of the recovery room should be kept at 75°F, and the relative humidity at 50% to meet federal guidelines, with six air changes per hour. As patients frequently leave the operating room mildly hypothermic, it is useful to have a blanket warmer or a heat shield in the PACU.

SUMMARY

In the PACU, the medical director in conjunction with the nurse manager sets standards of care, criteria for admission and discharge, and quality assurance. The nurse manager directs, coordinates, trains, evaluates, delegates, plans, and budgets for the unit. The essential role of the staff nurse in assessing, developing, and implementing a nursing care plan and then evaluating the care that she has rendered with or without the help of nursing assistants is covered as well as the requirements for training of the staff nurse and ancillary personnel. Lastly, the physical layout characteristics of a PACU are described.

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The Treatment of Acute Postoperative Pain in the Postanesthesia Care Unit

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At this end of the twentieth century we have finally begun to distinguish clearly the difference between acute and chronic pain. We have even begun to note that there are differences among various acute pain syndromes and to note that there may be both acute and chronic aspects of a given pain syndrome, so that we are seeing more work on the treatment of acute as opposed to chronic cancer pain, for example. We are noting such pain syndromes as are characterized by acute recurring or acute relapsing pain such as the pain of sickle cell crisis and some other medical conditions. Postoperative pain is another subclass of acute pain and requires its own set of approaches. We now propose to subdivide even further so that we can easily talk about the early versus late treatment of postoperative pain, because the approaches and problems may often be different in the same patient depending on the operation, anesthetic technique, plan for further postoperative pain management and, of course, individual needs of the patient.

In this article we will attempt to delineate some sound principles of early postoperative pain management, specifically management of pain in the postanesthesia care unit (PACU). Much of what is here presented is not specifically delineated in the literature as appropriate to PACU care and represents the distillation of literature material,

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extension and revisions of more general concepts in acute pain management, our own work, and the accumulated clinical experience of the Acute Pain Management Service of our institution.

During this early period in the patient's postoperative course, the anesthesiologist has a unique opportunity to influence the overall quality of patient care and the patient's satisfaction with the care given in the postoperative period. Needless to say, this treatment must begin with a proper understanding of the source of the patient's complaint, proceed through the development of a rational plan, and culminate in a satisfactory treatment of the problem, which we hope leads to its resolution, the appropriate relief of pain in the postoperative period. A smooth course in the management of postoperative pain ideally begins in the preoperative period and may begin with the anesthetic, but it must begin by the time of the first intervention for pain in the PACU.

A PRELIMINARY WORD ABOUT CHILDREN

It is not possible here to devote the amount of space that would be appropriate to the particular problems of treating early postoperative pain in children, especially infants and neonates. Much of what is said regarding adults, particularly with regard to *attitudes* surrounding the treatment of pain, applies doubly in children. Children *do* experience serious and undertreated postoperative pain just as do adults. Simple solutions ("Just let her mother hold her") and the old wisdom ("He won't remember the pain") must be discarded at the outset.[14] Efforts at understanding and quantifying children's pain require application of sound principles, but fortunately the pharmacology of early pain management is not so dramatically different in children over approximately 6 years of age that the principles laid out here cannot be applied. With children as with adults, however, to treat pain successfully one must suspend disbelief in the patient's report. When the patient says "I hurt," it is true, regardless of whether the observer feels there should or should not be the amount of pain the patient reports.

THE CONCEPTUAL FRAMEWORK

One of the most difficult of all attitudinal obstacles to the appropriate management of postoperative pain is the concept of the "pain well," the source from which one draws however much pain is appropriate when one has an operation. Thus, the unspoken dictum of the pain well concept would instruct us that a patient having a gastrectomy always has more pain than a patient having a skin operation.

In fact, there is a seed of truth in this concept. The definition of pain—a sensory and emotional experience associated with actual or potential tissue damage or which is described in terms of such damage[16]—certainly can include the idea that the more potent the stimulus, the more intense is likely to be the experience associated with it. Let us add to our armamentarium of definitions the following:

—Noxious stimulus: Any stimulus the intensity of which is actually or potentially inimical to the integrity of tissue.
—Nociception: The process of detection and signalling the presence of a noxious stimulus.
—Suffering: The reaction of the organism to the experience of pain.
—Pain-related behavior: The manifest actions of the organism that are the observable consequences of pain and suffering.

The representation that best characterizes the relationship among these entities is the famous "multifaceted model for pain" of Loeser[13] (Fig. 1), in which we see that *noxious stimulus* is not even present. It matters not, however, because to the observer all that is seen is the "pain behavior" and the relative size of the inner circles is of no importance. Certainly a more extensive surgical dissection will lead to the release of greater quantities of the various components of the inflammatory response and other pain-causing substances[5] (algogens). We would expect, therefore, that the patient who has a more extensively tissue-damaging operation will draw from the pain well using a larger bucket, but we have all had the experience of two patients who have undergone similar operations with similar anesthetics, one of whom seems to experience great pain whereas the other appears to have little pain. Often this difference is described in pejorative terms applied to the patient who seems to have the greater pain, but it is fully understandable if one understands the definitions and the *experiential* nature of pain. There is nothing wrong with the concept of the "noxious stimulus well" or even the "nociception well," but pain is much more than the sum of its physiological antecedent parts.

Nociception is modulated by many things including ethnocultural backgrounds and learned behaviors (Fig. 2)[1] as well as the meaning of the particular experience for the individual. The triad of locus of control, meaning, and mind-set all come to influence the amount of pain a patient experiences in a given postsurgical setting in a way which is well illustrated in Figure 3. It is the result of this combination of modulating factors, some ameliorating and some augmenting, which can lead to confusing and apparently paradoxical situations in early postoperative pain control. Their existence must be accounted for in the process of making an appropriate judgement about a method of treatment. It is understandable that a patient who has had a skin operation for excision of a potentially metastatic malignant melanoma

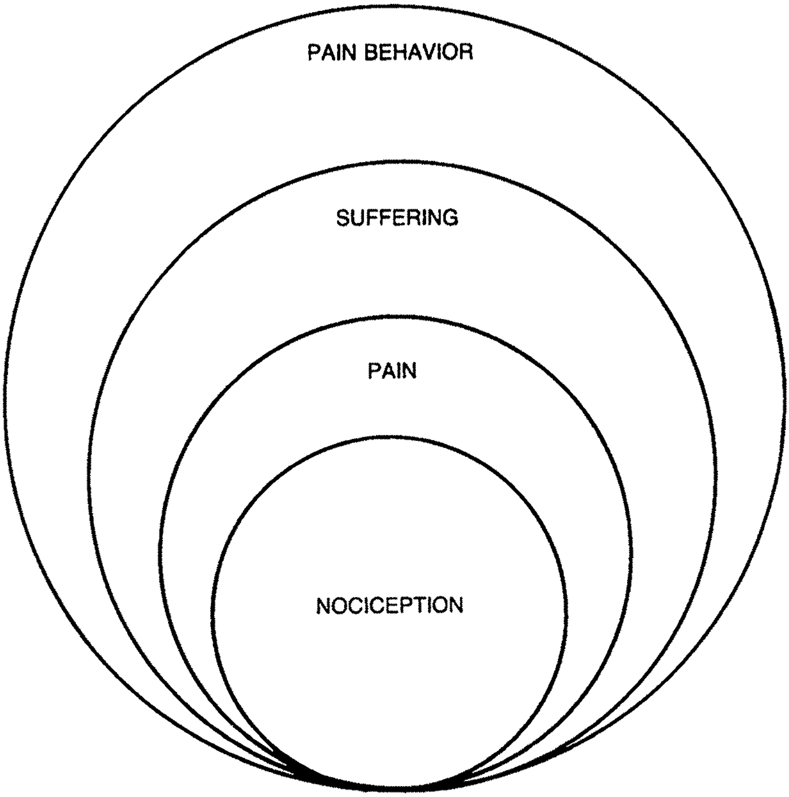

Figure 1. The multifaceted model of pain as proposed by Loeser. Notice that the relative size of the circles is unimportant to the model. The only observable sign is the pain-related behavior. (*From* Loeser J: Concepts of pain. *In* Stanton-Hicks M, Boas R, (eds): Chronic Low Back Pain. New York, Raven, 1984, p 146, with permission.)

may experience more pain than a similar patient who has had the same operation for cosmetic purposes, simply because of the meaning of the situation to the two patients.[4] In general, a single approach to treatment will not be so successful as the application, for example, of both pharmacologic and nonpharmacologic approaches, and any approach that fails to account for individual patient variability in all aspects of the pain experience will result in a patchwork of treatment results.

We may say, then, that the correct treatment of postoperative pain throughout the time of the postoperative course, but especially in the early period immediately following surgery, is founded on the following four principles: (1) correct diagnosis of the source and magnitude of nociception; (2) understanding of the relationship of ongoing nociception and other components of pain including anxiety, ethnocul-

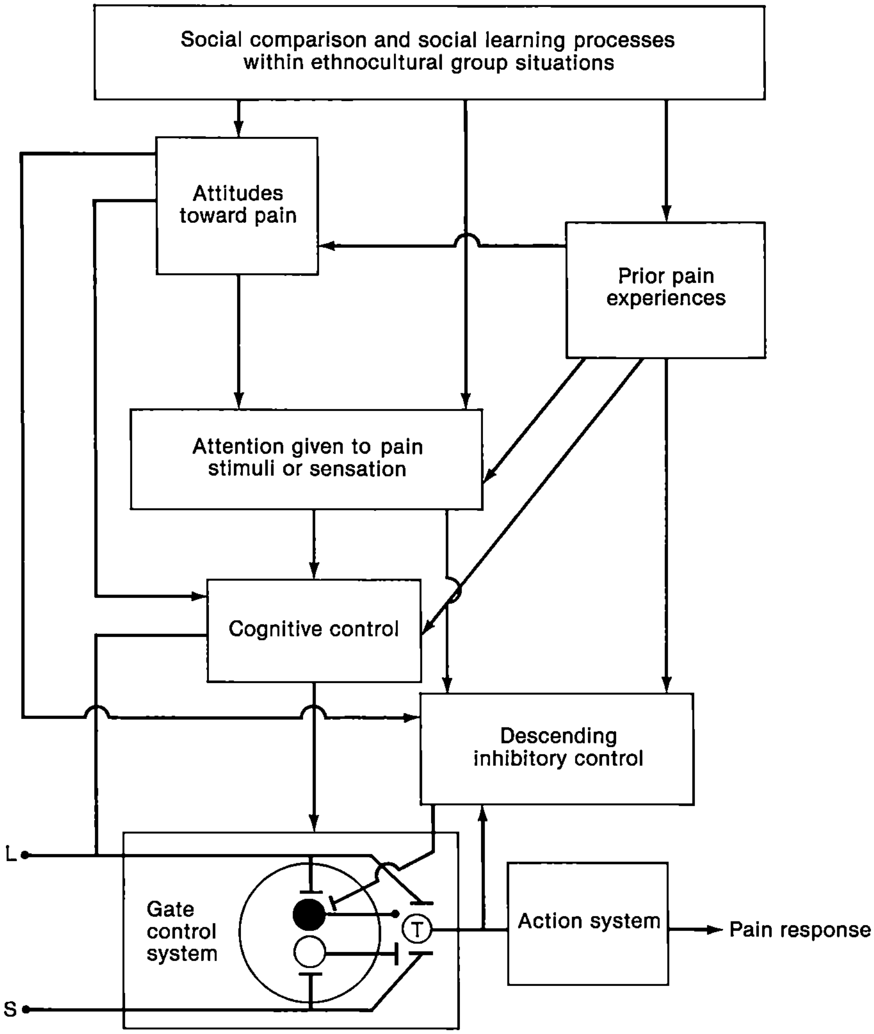
Social comparison and social learning processes within ethnocultural group situations
Attitudes toward pain
Prior pain experiences
Attention given to pain stimuli or sensation
Cognitive control
Descending inhibitory control
L
Gate control system
T
Action system
Pain response
S

Figure 2. The biocultural model demonstrates the multiple levels of modulation that come to bear on the pain experience through ethnic and cultural background, learned behaviors, and so forth. (*Adapted from* Bates MS: Ethnicity and pain—a biocultural model. Soc Sci Med 24:47–50, 1987.)

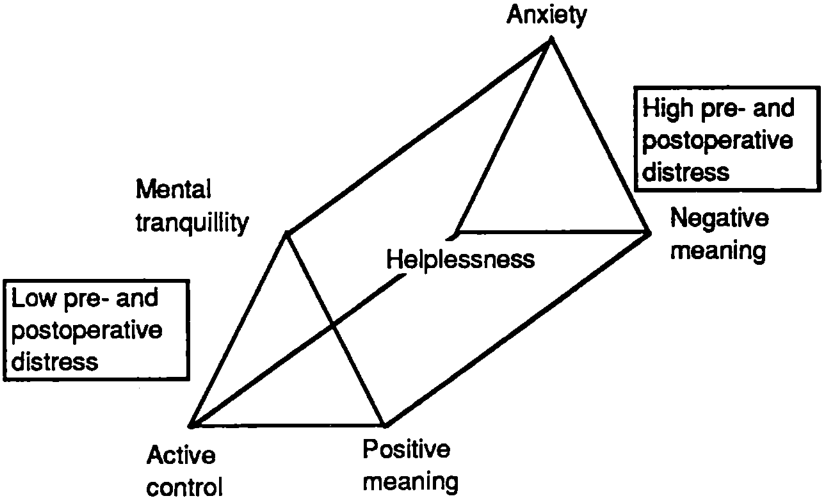

Figure 3. The triad of influences that can shape the character of a surgical experience for patients. (*Adapted from* Smith D, Covino B: Acute Pain. Boston, Butterworths, 1988; with permission.)

tural components, meaning, prior experience, and so forth; (3) treatment by establishment and maintenance of drug levels at active sites to achieve and maintain analgesia and anxiolysis as appropriate; and (4) continued re-evaluation of the therapy and refinement of the approach.

THE SOURCES OF SURGICALLY INDUCED PAIN

Incisional Pain

Although the activation of nociceptive neurons in the skin is known to be the final common peripheral pathway for nociception, direct stimulation of the nerve endings is rarely the cause of acute postoperative pain. Rather, the disruption of the intact integument causes release of chemicals (algogens) that secondarily interact with receptor sites to cause neural stimulation. Such chemicals include potassium ions, acetylcholine, histamine, peptides such as bradykinin, and an array of prostaglandins.[5]

Substance P and somatostatin, although not effective in causing pain when applied locally, may be released by local nociceptive neurons to increase the response to other algogens. The prostaglandins seem to function synergistically in the stimulation/sensitization of nociceptors, but are created and released locally rather than intraneuronally.

Additional stimulation for nociception can occur through the recruitment of the sympathetic nervous system. This is well known in the group of syndromes collectively known as ***sympathetically maintained pain*** (in an older taxonomy these pain states were known as the reflex sympathetic dystrophies such as causalgia, shoulder-hand syndrome, and Sudek's atrophy), but the contribution of the sympathetic nervous system as a cause of acute postsurgical nociception in most cases is small. Generalized sympathetic activity, on the other hand, is a well-known marker for pain. This is discussed later.

Localized Muscle Spasm

A natural defense mechanism in the face of nociception is guarding. This is localized muscle contraction that occurs involuntarily to prevent motion of an injured part. A teleological argument states that this tends to reduce continued nociception through reduction of the noxious stimulus. Although localized muscle spasm is only occasionally a pain problem in peripheral limb injuries, it is commonly contributory to nociception seen in intrathoracic and intra-abdominal surgery. This muscular contraction is usually only slightly painful, but when frank spasms occur, these spasmodic uncontrolled muscle contractions may become so intense as to overshadow the incisional nociception and may be refractory to treatment with narcotics. Therapy must be directed toward breaking the spasm-pain-spasm cycle. Narcotic analgesics will help to treat the pain, and a muscle relaxant may be added. This is probably one of the most common uses for benzodiazepines in the immediate postoperative period.

Positioning Pain

The positioning of a patient during surgery or during the immediate postoperative care may tend to aggravate muscle spasm pain. This can usually be overcome by repositioning the patient or even massaging the affected muscle (a technique that most PACU nurses use quite effectively). Pain related to pressure points that occur during surgery may be related to some degree of ischemia of the skin and underlying tissues. These pressure points may lead to aching in areas of the skin that have received bony pressure during prolonged procedures and may be prevented by proper padding. Once they have occurred, however, it is important to reposition and decompress the affected part to prevent progression of an ischemic injury.

More significant, however, are neural deficits caused by positioning. These deficits are created by localized pressure on nerves as they pass between bone and hard surfaces, such as the stirrups for lithotomy position, or by incorrect positioning of the extremities, for instance, leading to brachial plexus stretch injuries. Neural injuries are best treated prophylactically rather than after the fact but, should they occur, the affected limb should be protected from further injury while any sensory or motor deficit continues. Documentation of any deficit as well as of the patient's pain complaint is, of course, essential.

Deep or Visceral Pain

Compared with somatic pain occurring after surgery, which can usually be identified as being located in a specific body part and with a well-defined character, visceral pain is usually ill-defined spatially and temporally, notifying the body that injury has taken place but little more. The character is most often described as "achy" but may be burning, crampy, and so forth. It may radiate or may be "referred" to another part of the body entirely (such as infrascapular or interscapular pain due to diaphragmatic irritation) and be diagnosed and treated incorrectly. Visceral pain is common after abdominal or thoracic surgery because of widespread peritoneal irritation and the poorly defined and nonsegmental nature of innervation of the peritonealized structures and the pleura. It occurs in concert with the segmental somatic types of pain described previously and may result solely from the presence of drains or chest tubes.

Other Treated or Untreated Surgical Problems

Pain may be associated with the surgery but not directly caused by it. Ischemia of a limb or bladder distention may lead to patient discomfort that would not be present if the patient had not had surgery. Patients undergoing staged procedures such as serial orthopedic procedures following trauma may complain of pain in an injured but nonoperated extremity following surgery to stabilize another part. Gallbladder pain may be precipitated by the use of narcotics as part of the anesthetic technique in a patient who has had no intra-abdominal surgery. The key to diagnosis of this sort of problem is a high index of suspicion and a willingness to explore an appropriate assessment of pain with the patient.

Intercurrent Problem Unrelated to the Surgical Procedure

Anesthesia. Pain complaints may be referable to the performance of the anesthetic and in some cases can represent significant complications of an anesthetic. Particular attention should be paid to the aforementioned pressure and stretch neural injuries. An acute neuropathy may be extremely painful and will sometimes be the principal complaint. Other common painful complications include intravenous sites because of painful venipuncture and fluid extravasation; sore throat from endotracheal tubes; corneal abrasions, and intraoral pathology such as dental, periodontal, or tongue injury. Additionally, the central psychological effects of some of the anesthetic agents (euphoria/dysphoria with narcotics; hallucinations with ketamine, nausea with all drugs) must be remembered as an alternative source of discomfort and, therefore, of complaint.

Other Medical Problems. Other medical problems, both known and unknown prior to surgery, may be another cause of postoperative pain. The stress of surgery may precipitate angina in the patient with pre-existing heart disease; gastrointestinal disease such as hiatus hernia or esophageal reflux may cause heartburn or an increase in ulcer pain, none of which need to be treated by the use of narcotics.

Previously undiagnosed disease may become apparent during the stress of surgery and anesthesia, and a good diagnostician will be required to resolve various questions in the all-too-frequent absence of a history in this changing, perioperative period.

Anxiety and Distress. There are at least three axes of patient discomfort, none of which specify "pain" or even nociception (see Fig. 3). We can label these anxiety-tranquility; helplessness-active control; and the negative-positive meaning of surgery. This has been partially discussed previously, but it bears repeating in this context of nonsurgical causes of pain.

Anxiety can lead to emotional discomfort and a physical image indistinguishable from acute nociception. Sympathetic tone is high, and heart rate, blood pressure, and pulse are all elevated. It is common to see this in patients who have not been well prepared for surgery, and unfortunately seems to be becoming more common in these days of no premedication in ambulatory and "same-day admissions" patients. Antecedent anxiety has a direct relationship to postoperative anxiety, and treatment of anxiety prior to surgery will help diminish postoperative anxiety.[22, 24, 27]

Control of the situation (as distinct from the situation in control of the individual) becomes a major issue for many patients. Freedom has been stripped from them; they find themselves confined to bed, usually unable to eat, and functionally incapacitated. Control can be regained for some patients by the use of active relaxation techniques to diminish pain or by the use of on-demand, patient-controlled analgesia.[27] Distraction may be of use, as well as proper discussion with the patient of the reasons for and results of surgery.

The negative or positive meaning of the surgery for the patient will directly affect the amount of pain perceived.[4] Surgery for reconstruction of an athlete's limb to allow him to return to a sport or a cesarean section to produce a healthy baby are both seen in much more favorable light than exploratory surgery for cancer or major disease. The emotional consequences of permanent loss of limb or function will have their own impact on the perception of pain in the immediate perioperative period.

Chronic Pain in Exacerbation. Patients with chronic pain syndromes come to surgery, not only for their pain syndrome but for other

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indications. Chronic pain may be exacerbated by the surgical procedure, by poor positioning (especially in the case of chronic low back pain), and by the psychological effects of surgery. Difficulties arise when therapy normally contraindicated for the patient with chronic pain (narcotic analgesics and local anesthetic blockade) become available for the patient's acute nociception state. This becomes more problematic several days after surgery, when the acute nociceptive state is resolved and the chronic pain syndrome predominates.

Planning ahead can be the best therapy for these patients. The realization that therapy will be administered and withdrawn, but that their acute pain syndrome will be adequately controlled, can make the situation more tolerable for these patients. Chronic pain patients still have chronic pain in the face of acute postoperative pain and the acute pain must be adequately treated as indicated. One must be careful not to confuse the patient's chronic pain complaints with complaints of acute postsurgical pain, however, even though there is a tendency for these patients to be somewhat inconsistent in their reports of pain. Appropriate evaluation is crucial, and involvement of the team that has been caring for the chronic pain problem is very helpful.

THE PROCESS OF EVALUATING ACUTE EARLY POSTSURGICAL PAIN

Regardless of how much preparation and planning has been carried out in the preoperative period, accurate assessment of the location, intensity, and character of the pain for which the patient requests treatment must be made. Only in this way can one arrive at a correct diagnosis of the cause of the pain. Only by repeating the same questions following treatment can the value (or lack thereof) of the intervention be gauged. Side effects of analgesia will become apparent as changes occur in sensorium during regression of residual effects of general anesthesia and with sensory and motor function following a regional block. The sedation that frequently accompanies narcotic administration will be easier to evaluate as general anesthetic effects regress, and the good and bad residual effects of any anesthetic technique can be gauged by this process of repeated questioning. Since it is unlikely that the correct solution will be achieved the very first try, refinements of therapy will likely be necessary. This is akin to the mathematical process of "successive approximations" to a solution.

Some Questions to Ask

Where Does It Hurt? Is the location of the pain appropriate for the surgery performed, the patient's current position, or is it in another

Table 1. *Guidelines for Action Depending on the Location and Quality of Early Postoperative Pain*

QUESTION	ANSWER	IMPLICATION	ACTION
Where does it hurt?	Operative site	Incisional pain, visceral pain, muscle spasm associated with operated body part, position problem	Direct result of surgery: think of definitive pain therapy
	Someplace distant from operative site	Anesthesia related, chronic pain, exacerbated, pre-existing medical problem, surgical problem unrelated to current procedure	Document, diagnose and treat specifically; consult if indicated
	Everywhere or don't know	Situational distress, pre-existing emotional problem	Think of nonpharmacologic or nonanalgesic solution
What does it feel like?	Sharp, cutting, pulling, aching	Likely related to surgery depending on answer to where does it hurt	Definitive pain therapy
	Buzzing, tingling, funny, numb, can't tell	May not be related to surgery depending on answer to where does it hurt	Document; look before you leap; go back to beginning and re-evaluate "someplace distant"

location entirely? The location of nociception (if any) will be informative. If nociception is not related to the surgical procedure, or to deep visceral or referred pain as applicable, then a secondary process must be investigated and treated, and any one of the several causes of postsurgical pain unrelated to the surgical procedure noted previously may be involved (Table 1).

What Does It Feel Like? The quality of the sensation is important. If it is sharp, it is probably direct nociception associated with the incision. Dull aching sensations, well responsive to narcotics, are typical of incisional pain arising from deeper tissue. Pulling or tugging is in keeping with the stitches in the wound and with some visceral stimulation. This aching pain may also be described as burning or cutting. Nausea, cramps, or an ill-defined sensation of not being well are typical with visceral and peritoneal irritation.

Buzzing, stinging, or a sensation of electricity indicate return of or abnormal neural function. These sensations are minimal following regional anesthesia. They are more indicative of neural compression

Table 2. *Techniques for Grading Pain Before and After Treatment*

Five Point Global Scale	None = 0 A little = 1 Some = 2 A lot = 3 The worst = 4
Verbal Quantitative	0 . . . . 5 . . . . 10 None Worst imaginable
Visual Pain Analog Scale (VPAS)	No pain Worst pain Place a mark on the line to show where your pain is now

and re-establishment of neural function when they occur in the acute postoperative setting. Painful dysesthesias, which may occur in peripheral neuropathies, may also occur as part of a pre-existing medical condition.

How Much Pain? Various forms of surgery hurt in various ways. It is important to remember that there will be different intensities of pain, depending on the operative site, previous therapies, and all the psychological effects mentioned previously.[24] It is advisable to have each patient try to quantify his or her pain level. The visual[11] or verbal analog scales are of use here (Table 2). It must be remembered that each patient serves as his or her own control, and response is best measured in changes from baseline values. A scale of "No pain/mild/moderate/severe" is at least getting the idea. A numerical scale without the visual analog can also be used effectively. One need only ask the patient to try to assign a "number from 0 to 10 to the pain: where 0 = no pain at all and 10 = the worst pain you could possibly imagine." This type of scale is not as accurate as a Visual Pain Analog Scale (VPAS) but requires only that there be some preoperative teaching that can occur even while putting in a patient's intravenous line preoperatively. An accurate assessment of changes in this pain level with therapy can then be made.[20]

How Sedated Is the Patient? Is the patient readily arousable? Is sedation a significant change from preoperative mental status? Is this sedation acceptable, or does the patient need to be monitored neurologically? Is analgesia obtained only when the patient is sedated to the point of unconsciousness? How acceptable is this level of consciousness both for the patient and for the persons taking care of the patient? How well attended will the patient be on the wards or in the intensive care unit?

Motor Function and Sensory Impairment. How much have the surgical procedure, regional blockade, and the analgesics given perioperatively interfered with homeostasis? Is the patient expected to be ambulatory postoperatively, or will he or she be confined to bed rest? What level of motor and sensory impairment is allowable in the perioperative period?

Previous Drug Therapy and Response. Review must be made of current and previous drug therapy. The patient who "didn't wake up for 3 days" following a previous anesthetic probably had a relative drug overdose, but it is important to rule out idiosyncratic reactions to drugs in the history. The patient who is on chronic narcotic analgesics before surgery can be expected to show some tolerance to narcotic analgesics and will require a higher dose. The exact degree of tolerance will be difficult to assess, but any patient who has been receiving a fixed narcotic dose for more than 48 hours before surgery will likely tolerate an increase of 25 to 50% in the dose without any difficulty. Cross tolerance almost certainly occurs between systemic narcotics and intraspinal narcotics.

Finally, when evaluating a patient postoperatively, it is necessary to see what methods of analgesia have been used intraoperatively. Were narcotics given? What dose? At what time? For what indication?

What was the anesthetic technique? Was it soundly based on narcotics? Was a primarily inhalational anesthetic supplemented with narcotics, or were narcotics avoided altogether? Was there a mixture of light general anesthesia and regional blockade? Were intraspinal narcotics administered?

Will there be an interaction between other anesthetic agents (barbiturates, benzodiazepines, and possibly local anesthetics) and narcotic analgesics?

Accounting for Residual Effects of Anesthesia: Good

The anesthetic may be designed to augment postoperative pain relief. The operating room is the perfect place to start systemic techniques, such as narcotic loading, and regional techniques, such as intraspinal narcotics and continuous local anesthetic infusions. Analgesia may be titrated in this setting, and the patient response observed under very controlled conditions.

Recommendations may then be made to the next practitioner taking care of the patient's analgesia, either the surgeon or the acute pain management service. Perioperative anxiety, expectations, worries, and drug responses can all be related to the primary care team who can then cooperate to maintain adequate postoperative analgesia for the patient.

Accounting for the Residual Effects of Anesthesia: Bad

Strategies can backfire if not continued postoperatively. Loading a patient with a narcotic will be of no long-term use if the continuing service does not continue with that narcotic schedule. Adequately functioning methods of neural blockade may be discontinued perioperatively by uninformed individuals.

Single-shot techniques of neural blockade can become problematic. The patient may be discharged with minimal pain during the recovery stay, all due to residual block. Once the residual neural block has worn off, however, the minimum effective analgesic concentration (MEAC) of an analgesic will not have been achieved. The well-planned postoperative analgesic strategy will not be effective. Patients who have been treated with certain butyrophenones such as droperidol may exhibit disorientation or even movement disorders that interfere with appropriate evaluation of their postoperative pain. Drowsiness from residual inhalational agent is not so problematic as at one time, but may be so in early PACU discharges.

THE TREATMENT PLAN

All treatment must begin with a plan that encompasses the benefits and shortcomings of the therapy that has been previously applied. Therefore, all plans for postoperative pain management begin by incorporating the results, good and bad, of the anesthetic (Table 3). The treatment plan may be extremely simple and include nothing but the

Table 3. *Early Postoperative Pain Management Plans*

Before induction	Begin narcotic that will form basis for postoperative plan as part of premedication
Very simple	Plan a narcotic/N_2O = O_2 anesthetic based on narcotic that will form basis for longer-term postoperative pain control
	Plan a regional anesthetic using an agent that will supply analgesia in postoperative period
	Plan an inhalational anesthetic using increasing narcotic supplement near end to begin postoperative pain control
Simple	Intravenous front loading in PACU for intramuscular maintenance (inpatients)
	Intravenous front loading for by mouth maintenance (inpatients and outpatients)
Complex	Intravenous front loading in PACU for continuous intravenous infusion of narcotic on ward; start infusion in PACU
	Intravenous front loading for PCA narcotic maintenance (with or without background infusion); start in PACU
	Choice of PCA interval dose and lockout time; involve acute pain management team if possible
Very complex	Continuous epidural infusion of local anesthetic
	Continuous peripheral regional infusion of local anesthetic (brachial/lumbar plexus)
	Continuous interpleural infusion of local anesthetic
	Intraspinal narcotic: single-shot intrathecal as part of subarachnoid block, intermittent epidural, continuous epidural, without local anesthetic and with local anesthetic

Abbreviation: PCA = Patient-controlled analgesia.

analgesia that extends into the postoperative period from residual effects of either a general or regional technique, or it may be as complex as the continuous use of an interpleural local anesthetic with ongoing monitoring by an acute pain management team. The planning must begin before the induction of anesthesia, however, because the early treatment or postoperative pain cannot be separated from either what has gone before or what is to come in the later postoperative period. Just as with the development of a good anesthetic plan, good postoperative pain management begins in the preoperative period. The best preoperative beginning is a discussion of the postoperative pain care options with the patient and with the surgeon. This avoids contradictory postoperative pain orders and mix-ups in communication that interfere with the best in patient care.

Approaches to Therapy

Preoperative assessment and interview of the patient is known to be the best source of decreased perioperative anxiety. Enhancing patient education will help minimize anxiety and the feelings of helplessness mentioned previously. Along with treating the emotional aspects, the problem of ongoing nociception and its treatment must be addressed. The next phase of therapy is to ensure that there is an adequate level of analgesic, either local anesthetic or narcotic, at the site of action that has been chosen for the analgesic technique.

The "gold standard" for postoperative analgesia has been, in the adult, 10 mg of morphine sulfate intramuscularly, every 3 to 4 hours intermittently as required to maintain analgesia.[26] Unfortunately, this gold standard all too often proves to be dross, and the reasons for failure of the regimen are many.[18] These failures may be avoided or prevented by proper prescribing practices and dosing schemes based on sound understanding of the pharmacodynamics and pharmacokinetics of the drugs and techniques employed.[8]

Prescriptions require definition of drug, dosage, mode of administration, frequency of administration, and conditions under which the drug is to be given. Major side effects of respiratory depression and over-sedation from narcotics are rarely seen in the acute postoperative setting if patients are carefully watched over a reasonable period of time (arbitrarily, twice the time to peak effect). Success lies with obtaining and maintaining a therapeutic level of drug that exceeds the MEAC, for both narcotic and non-narcotic medications.

Very Simple Therapeutic Techniques

The easiest approach to postoperative analgesia is to incorporate the use of narcotics intraoperatively for postoperative analgesia. This can be accomplished in several ways: Use of a N_2O-O_2/narcotic/relaxant technique, with or without potent inhalational supplementation; use of inhalational anesthesia with narcotic supplementation, either throughout the case or at the end of the case; or use of regional anesthesia with or without general anesthesia with intraspinal narcotics given before the block recedes or the patient emerges from general anesthesia.

Unless otherwise contraindicated, it makes the most sense to use a single analgesic agent throughout the perioperative period. For example, if morphine is to be used postoperatively, then morphine used intraoperatively can be counted as part of the drug loading and maintenance schedule.

In particular, it makes little sense to use one narcotic, such as fentanyl, in the early postoperative setting, when the patient is to be maintained on another drug such as meperidine, in the later postoperative setting. If a short-acting narcotic is indicated as part of the anesthetic technique, then loading the definitive analgesic early in the postoperative period should be the rule. In general, a little planning prior to the induction of anesthesia can make a significant difference in the emergence of the patient from his or her anesthetic. Strategies must then be developed to mesh early postoperative pain management in the PACU with what is to occur in the later postoperative period.

Simple Therapeutic Techniques

A simply strategy for obtaining and maintaining pain relief is to obtain analgesia by front-loading techniques perioperatively and maintain drug levels either orally or through intermittent deposition of analgesic medication intramuscularly or subcutaneously. The depot drugs will be slowly absorbed, and if the repeat injections are properly timed, their absorption should approximate the clearance of the drug from the blood stream and leave minimal variances in blood analgesic level. When choosing this approach (1) *do not begin until and unless analgesia has been achieved by intravenous front loading,* and (2) *do not begin until circulatory physiology is normal at the depot site.* Several factors must be considered when choosing a drug for postoperative analgesia. What will the intensity of the pain be? What strength of drug will be needed and what drug will be sufficient without excessive side effects? This is often not the choice of the anesthesiologist caring for the patient in PACU but will have been decided by the surgical team. Regardless of how the choice of analgesic drug is made or what that choice is, front load the narcotic with which analgesia will be provided for the later postoperative period. A schedule of drug choices and loading techniques is offered in Table 4. Caveats are noted as applicable.

Some Factors Affecting Drug Choice

Weak Narcotic: Codeine. Codeine is a relatively weak narcotic that is sufficient for mild to moderate pain. It can be administered

Table 4. *Guidelines for Front Loading Intravenous Analgesics*

DRUG	TOTAL FRONT-LOAD DOSE	INCREMENTS	CAUTIONS
Morphine	0.08–0.12 mg/kg	0.03 mg/kg every 10 min	Histamine effects; nausea; biliary colic; reduce dose for elderly
Meperidine	1.0–1.5 mg/kg	0.30 mg/kg every 10 min	Reduce dose or change drug for impaired renal function
Codeine	0.5–1.0 mg/kg	⅓ total every 15 min	Nausea
Methadone	0.08–0.12 mg/kg	0.03 mg/kg every 15 min	Do not administer maintenance dose after analgesia achieved; accumulation; sedation
Levorphanol	0.02 mg/kg	50–75 µg/kg every 15 min	Similar to methadone
Hydromorphone	0.02 mg/kg	25–50 µg/kg every 10 min	Similar to morphine
Pentazocine	0.5–1.0 mg/kg	½ total every 15 min	Psychomimetic effects; may cause withdrawal in narcotic-dependent patients
Nalbuphine	0.08–0.15 mg/kg	0.03 mg/kg every 10 min	Less psychomimetic effect than pentazocine; sedation
Butorphanol	0.02–0.04 mg/kg	0.01 mg/kg every 10 min	Sedation; psychomimetic effects like nalbuphine
Buprenorphine	up to 0.2 mg/kg	¼ total every 10 min	Long-acting like methadone, levorphanol; may precipitate withdrawal in narcotic-dependent patients; safe to give subcutaneous maintenance after analgesia, different from methadone

subcutaneously or intravenously, but is usually given orally. Codeine is associated with considerable nausea and vomiting. Ventilatory depression may occur relatively late; do not administer codeine phosphate more frequently than every 15 minutes. The relative poor bioavailability of codeine administered orally must be remembered. (Equianalgesic potency is 120 to 200 mg of codeine phosphate orally equals 10 mg MSO_4 intramuscularly).

Codeine is indicated as a narcotic analgesic for the patient who will return to oral intake quickly (within 24 hours) or for the ambulatory patient.

Strong Narcotic. In the acute postoperative setting, the class II narcotics are rarely ineffective if enough drug is given. They are equivalent in terms of ability to produce analgesia and the side effects of respiratory depression, tolerance, and dependance (and addiction). They differ in potency, duration, metabolism, and other side effects (predominantly nausea, vomiting, urinary retention, and pruritus). Rarely is drug choice a problem in obtaining adequate analgesia.

Morphine. The ventilatory depression associated with morphine peaks at 10 minutes after intravenous administration. Morphine should not be given incrementally more frequently than every 10 minutes. Do not administer additional intravenous doses beyond the achievement of adequate analgesia. At that time, the first maintenance dose may be given parenterally.

Meperidine. Nausea and vomiting are common with intravenous meperidine. Ventilatory depression peaks 5 to 10 minutes after administration of intravenous meperidine. Incremental intravenous doses should not be given more frequently than every 10 minutes. Do not administer additional intravenous doses beyond the achievement of adequate analgesia. Use intravenous meperidine with caution in patients with a history of seizure disorder, as the first metabolite, desmethylmeperidine (normeperidine), is a central nervous system stimulant with a half-life of 12 to 17 hours. Accumulation of this metabolite is rarely a problem but can occur, particularly in the patient with renal failure if adequate attention is not paid to total hourly consumption of drug over a 24-hour period, which should not exceed 1500 mg in patients with normal renal function under most circumstances.

Other Potent Narcotics. Drugs such as dihydromorphone (Dilaudid), levorphanol (Levodromoran), and methadone (Dolophine) may well be chosen for routine postoperative care for any number of reasons including previous good experience of the patient with that drug, familiarity of the team caring for the patient on that drug, preoperative use of the drug by the patient for ongoing pain problems, and so forth. Whatever the reason, if one of these potent narcotics is to be used to provide postoperative analgesia, that drug should be front loaded in the PACU and the patient should receive the first intramuscular or subcutaneous dose that has been prescribed for maintenance as described previously. The sole exception to that guideline is methadone, which potentially has such a long circulating half-life that the therapeutic window for the drug could well be exceeded during absorption from a tissue depot. Methadone should be front loaded exactly like morphine. Stop administering methadone when analgesia is achieved and do not give additional drug until the patient requests additional medication. This will likely be at about 3.6 hours. A maintenance schedule of every 8 hours is then feasible.

Mixed Activity Drugs. These are a group of drugs that are gaining some popularity in postoperative pain control. They include both the narcotic agonist-antagonist drugs and the partial narcotic agonists. Among the assortment of these drugs available worldwide, nalbuphine, butorphanol, and pentazocine are grouped in the former category and are available in the United States, whereas among the latter category of drugs only buprenorphine is available. The use of these agents is very similar to the use of pure narcotic agonists in that they can be front loaded by intravenous administration and maintained on a schedule of parenteral administration consistent with their pharmacology. Advantages of these drugs seem to lie in their dose-response relationships, which show a ceiling on their ability to depress ventilatory response to CO_2. Relative disadvantages exist in their tendency to reverse their own analgesia at high doses, and their ability to induce acute abstinence syndrome in narcotic-dependent individuals. Only pentazocine is available in a nonparenteral formulation in the United States but buprenorphine (sublingual) is currently being studied. Except for buprenorphine, these drugs tend to be limited in their clinical application by their tendency to produce dizziness, dysphoria, and an array of psychomimetic effects such as hallucination. The frequency is in the order pentazocine > butorphanol > nalbuphine. All may produce significant sedation when given intravenously.

Route of Administration for Systemic Narcotic. It has been and usually still is customary to give narcotics intramuscularly on the wards during efforts to gain analgesia. In the PACU, drugs should be given intravenously. Rectal and (rarely) oral administration may be acceptable under the circumstance of no intravenous access, but specifically one should avoid subcutaneous and intramuscular routes of administration.

With the change in cutaneous and intramuscular blood flow associated with varying degrees of postoperative hypothermia, the absorption from intramuscular or subcutaneous sites becomes unpredictable. The same would be true of a drug such as transdermal nitroglycerine. Drug administration intravenously is immediately available from blood to the target tissue, and there is the fastest onset of analgesia (within 15 minutes for morphine and meperidine). There is no depot from whence the drug may return to the circulation as the patient begins to rewarm, as opposed to subcutaneous and intramuscular caches. The sole exception to this rule may be for the patient who is to be discharged on routine intramuscular injections on the postoperative ward. Under those circumstances, intravenous drug can be administered to effect, and then the intramuscular dose given so that the circulating level of drug stays in the therapeutic range after transfer to the floor.

Dosage Intervals for Systemic Narcotic. Effective analgesia can be maintained only when the medication is repeated while the previous dose is still effective. The peak effect is just that. The next dose should be administered quickly after the time of peak effect so that there may be a cumulative effect leading toward MEAC during the period of titration with small doses to the desired effect.

Non-narcotics

Nonsteroidal Anti-inflammatory Drugs. The nonsteroidal anti-inflammatory drugs (NSAIDs) are generally insufficient analgesics for severe postoperative pain. Through their peripheral effects on prostaglandins, however, they decrease peripheral nociception. They are not effective against visceral pain, but are additive with the narcotics against noxious somatic stimuli. They are particularly effective when inflammation and trauma are present.[12] With the exception of indomethacin, which is available as a rectal suppository, they are available only in oral formulation. This requires that the patient be tolerating oral intake before they can be reliably administered. However, they may be administered preoperatively and their administration continued postoperatively as soon as oral intake is allowed. Some, such as naproxen (Naprosyn) and diflunisal (Dolobid), have a long enough half-life to give them on the morning and the night of surgery.

These medications have a variety of side effects. Among them, all but choline magnesium trisalicylate (Trilisate) and salsalate (Disalcid) decrease platelet function in a reversible manner. All but choline magnesium salicylate have been associated with gastric irritation. These drugs have been known to cause bronchospasm.

Transcutaneous Nerve Stimulation. Transcutaneous nerve stimulation (TENS) was originally thought to be of use in decreasing the acute incisional pain after such surgeries as thoracotomy and cholecystectomy[7] and cesarean section.[23] Further studies have shown a decrease in pain ratings postoperatively, but no change in narcotic usage with the use of TENS or with a sham TENS control. Additionally, there was no significant difference between control patients and those with TENS on postoperative respiratory function. The routine postoperative use of TENS in most patients cannot be recommended.

Complex Therapeutic Techniques

Both of the following techniques, timed parenteral administration and continuous intravenous infusion of narcotics, are applicable primarily for patients who will have prolonged stays in PACUs. Although they are sophisticated, they are quite easy to carry out under usual monitoring conditions on the wards, particularly if patients have been in the PACU under observation for about three drug half-lives and assurance can be given about the patient's response to the technique. Any patient who has been receiving continuous or timed narcotic preoperatively should have it re-instituted postoperatively (preferably maintained intraoperatively) so as to avoid narcotic withdrawal syndrome as well as to maintain good analgesia.

Timed Parenteral Administration of Narcotic. One of the most common failures of drug therapy occurs through of the use of medications on an "as needed" basis. Sliding dosage and administration scales were thought to allow flexibility in treating pain. The basis of as-needed administration is that the indication for analgesic therapy occurs before the drug can be administered; this is the first stop in the control cycle. Following that, the patient is evaluated by the nurse, who then finds the narcotics keys if she or he thinks the patient needs medication, then administers a dose virtually independent of that ordered, and (by now) at a different time than when requested. By the time the drug is absorbed and blood and tissue levels increase to therapeutic range, the patient has been without adequate analgesia for times up to 1 hour in duration.

This can all be avoided by the use of time-contingent dosing. The dose of medication is administered by the clock, rather than on demand, so that the new dose of drug will have time to be absorbed before the remaining (previous) drug administration has time to fall beneath the therapeutic level.

The duration of action of most of the potent narcotic medications (or the pharmacodynamic half-life) is approximately 3 hours. Medications should be given on this schedule to maintain drug levels at or above the MEAC for the duration until the next dose has time to become effective. The dose administered in this time period must be sufficient to maintain analgesic level above the MEAC for the duration of the time period, but low enough to avoid excessive sedation and respiratory depression. If a dose sufficient to cover the dose interval is too large to avoid these side effects, the dose must be reduced and given more frequently. The pharmacokinetics of the patient are better served in this manner, as the delay to analgesic effect is minimized. Similarly, the dose interval should remain constant to allow the full duration of the action of the drug; the interval should be consistent with the pharmacokinetic half-life, generally 3 hours.

Continuous Intravenous Infusion of Narcotic. The limit of the previously mentioned dosing scheme would be very small amounts of narcotic administered very frequently, thus obtaining second-to-second matching of drug elimination with drug administration. To achieve this end, narcotics may be infused in a continuous manner. After an initial priming dose (which has been infused or given as incremental boluses) to create analgesia, an estimate is made of the drug clearance. This amount of drug is then continuously administered intravenously to maintain a relatively constant amount of drug in the bloodstream.

The continuous infusion technique is not dangerous provided some simple principles are followed. Pharmacokinetics tells us that it takes three half-lives to reach 90% of a new steady-state level with the continuous infusion of any medication, and approximately five half-lives to reach true steady state. Since the half-life of morphine is 2 to 3 hours, it requires 6 to 9 hours to approach the plateau of accumulation. This should be plenty of time to note that a patient is becoming too somnolent (accumulating unnecessary analgesic) or not receiving enough narcotic (breakthrough pain).

Since the elimination kinetics of morphine are first-order (constant *fraction* per unit time), the amount of drug that needs to be administered continuously will be a function of the amount of drug required to achieve analgesia. The rate of infusion can then be estimated by estimating clearance. An estimate can be made of the clearance by assuming that in 3 hours, half of the originally administered dose will be metabolized (this is a slight underestimate due to tissue binding and distribution to other tissues, but will do as an approximation). This amount must be administered over those 3 hours to cover drug metabolism. Thus, for instance, if the original analgesic front load were 15 mg, then 2.5 mg/hour would slightly underestimate the patient's requirement (15 mg ÷ 2 = 7.5 mg ÷ 3 hours = 2.5 mg/hour).

If underdosing becomes a problem, the patient will need to be rebolused intravenously until comfortable. If they have become overly somnolent, then stopping the infusion for a while to allow symptoms to regress as the level falls toward a new steady state would be effective therapy.

Naloxone is, of course, a specific narcotic antagonist that will resolve narcotic-induced somnolence or respiratory depression immediately. This drug must be administered with caution to avoid reversing analgesia and producing an analgesic state. In addition, the half-life of a single dose of naloxone is about 20 minutes and renarcotization can occur after that.

Patient-Controlled Analgesia. A hybrid of the continuous infusion and intermittent as-needed medication, the patient-controlled analgesia strategy allows the patient to receive narcotics as needed on demand (similar to an as-needed schedule) but in very small increments and frequently (as with continuous infusion). It is best to think of it as a variable infusion controlled by the patient. Within the parameters designated by the prescribing physician, the patient can give small, repeated doses of narcotic analgesic as his pain returns, thus maintaining a relatively uniform level of analgesic, just above MEAC.

This strategy is superior to the continuous infusion of narcotics in two ways. First, it reduces the need for monitoring of the patient as the sedated patient is unable to administer analgesic inappropriately; accumulation does not occur. The patient's pharmacokinetics are more closely matched with the patient being the feedback controller. Second, the problem of the shifting baseline for analgesia is removed. The patient will administer less narcotic as less is needed.

Most of today's patient-controlled analgesia (PCA) devices have a mode for the continuous infusion of narcotic (the "basal" rate). The original intent of the basal rate was to decrease the number of demands that a patient would have to make during periods of sleep or deep rest. This basal infusion may become excessive for the patient over time and lead to overdosage in a manner similar to that of overdosage seen with continuous infusion of narcotics (see previous discussion). The treatment of this problem is to remove the basal infusion.

A general rule should be kept in mind: PCA alone without a frontload does not make anyone comfortable. It will keep someone comfortable by maintaining a narcotic level just above MEAC. It is extremely difficult for someone with a significant change in analgesic requirement to increase his level of narcotic appreciably if the prescription has been properly written.

Very Complex Therapeutic Techniques

More complex pain strategies have evolved as our knowledge and interest in the therapy of postoperative pain continues to grow. These strategies rely on the applications of narcotics to the spinal cord where, at the substantia gelatinosa, they may decrease ascending fiber firing and thereby decrease nociception. Such use of narcotics is called *intraspinal* as that is the mechanism of action.

Four modes of intraspinal narcotic therapy have been used successfully at various institutions: single-shot techniques, commonly associated with intrathecal narcotics; intermittent bolus injections, the "traditional" method of administration of morphine intraspinally (usually via the epidural space); and continuous infusion of narcotics, with or without local anesthetics.

Other strategies have involved the use of continuous regional analgesics with local anesthetic agents alone by infusions into a regional neural plexus. The newest of the regional techniques is probably interpleural analgesia (called intrapleural by some authors).

Intraspinal Narcotics. Since the late 1970s, intraspinal narcotics have revolutionized modes of analgesic therapy. Multiple techniques have been used and are well described elsewhere. The decision to use intraspinal narcotics rests on the answers to the following: Will the patient accept regional anesthesia/analgesia? Is the patient a candidate for regional anesthesia/analgesia? Is the surgical site amenable to intraspinal narcotics? Surgery above the second cervical dermatome (the forehead and face) involves the cranial nerves. This area is not innervated by the spinal cord and therefore intraspinal narcotics are not indicated.

There is little evidence that sites of nociception more than a few dermatomes apart will receive adequate doses of intraspinal narcotics (particularly when epidurally applied). Conversely, however, it appears that it is possible to achieve good analgesia when the site of epidural injection is several dermatomal segments from the operative site, for instance, lumbar epidural catheters for analgesia for thoracotomy pain. This is particularly true for the use of epidural morphine, and less so for the use of the more lipid soluble agents.

Minor side effects of intraspinal narcotics include pruritus, nausea and vomiting, and urinary retention. Except for urinary retention, infusions of naloxone, 1 to 2 μg/kg/hour intravenously, will reverse these side effects. Urinary retention appears to be reversible only when the analgesia is reversed, at doses of 20 to 30 μg/kg/hour. Nalbuphine has been used in a similar fashion for the prevention of side effects.[2]

Intermittent Dosing. In the intermittent scheme of dosing, a large amount of narcotic is administered every 8 to 12 hours into the epidural space.[20] Analgesia from this dose will range from 8 to 24 hours, and sometimes even longer. The advantage of this dose schedule is that it is mechanically simpler (no infusion pump necessary) and decreases the manpower requirement for injecting catheters. The disadvantage is that patients may break through and require additional doses of medication. Additionally, the use of bolus dosing increases the possibility that, if the catheter has migrated into the subarachnoid space, the full injection of narcotic may be placed intrathecally, potentially resulting in profound respiratory depression. This latter problem is readily and consistently avoided by careful aspiration of the catheter prior to injection.

Continuous Infusion. *Without Local Anesthetic.* Continuous infusions of epidurals have been used to increase the efficacy of analgesia. The advantage is that less manpower is required for epidural injections. The disadvantages include the requirement for an infusion device. Fentanyl is most commonly used for this procedure. A concentration of 10 μg/mL infused as 10 mL/hour via the epidural space seems to be as effective as 5 mg of epidural morphine every 12 hours.

Because of its increased lipid solubility, fentanyl has the theoretical advantage of being able to produce a more segmental analgesia. Although this may be a problem in patients who have catheter sites distant from the operative site, it allows a more controlled level of analgesia in patients for whom other nociceptive sites must be monitored. Since it is a continuous infusion, it is more readily titratable than is the same drug given by the intermittent bolus technique.

Supplement to Narcotics with Local Anesthetic. Local anesthetics have been used in conjunction with epidural narcotics to increase analgesia. It is not clear that 0.125% bupivacaine with fentanyl, 10 μg/mL, is a better analgesic than bupivacaine alone, or that the effects of fentanyl are not due to its systemic absorption.

Local anesthetics clearly have a place in treating the acute nociceptive state while waiting for the epidural narcotic to become effective. Additionally, we have seen many patients who have had no effect from epidural narcotics until very low dose local anesthetic blockade, in the sympathetic blocking range for bupivacaine, has been achieved. This may result because deep visceral and peritoneal nociception is not readily blocked by epidural narcotics. The central pain pathways involved in nociception of peritoneal and pleural inflammation and irritation are not all via the substantia gelatinosa; it should not be surprising that pain arising from these structures is ineffectually treated by intraspinal narcotics.

Continuous Regional Analgesia

Epidural. As mentioned previously, epidurals can also be used for truly continuous blockade of nerves by the infusion of local anesthetic solutions. If this is to be done, as mentioned, the drug *must* be brought to the effective concentration (the critical concentration) before analgesia will occur. The infusion of local anesthetic then maintains that concentration at the nerves, predominantly in the epidural space.

An estimate must be made for the continuous infusion. The infusion rate is related to the mass of drug that is required for an intermittent dosing technique. Generally, the concentration for continuous infusion must be lower than that for an intermittent bolus; the volume of solution must be higher, but the net effect is that the mass of drug (concentration times volume) is the same. If this guideline is not followed, the block will contract, become less effective at border-zone dermatomes, and will increase in intensity.[19]

Generally, for surgical analgesia, concentrations of 0.125% (eighth per cent) to 0.2% of bupivacaine are effective. If used with narcotic (see previous discussion), doses as low as 0.0625% (1/16th per cent) may be used with good results. Infusion rates of 10 to 20 mL/hour are not uncommon.

Brachial/Lumbar Plexus. Continuous brachial plexus blocks have come into use in recent years. Although they are highly effective for postoperative analgesia,[19] they have a real place for continued sympathetic blockade of an extremity after major vascular repairs, skin grafts, and for treatment of sympathetically maintained pain.

A catheter is placed within the perivascular sheath and secured. The interscalene, subclavian perivascular, and axillary approaches using regular intravenous catheters have been described. An approach via the axillary space, with placement of a "through-the-needle" catheter has been described[19] in a manner similar to the placement of a continuous epidural block. This can be achieved in the femoral perivascular space, also. Location of the correct space for catheter placement by use of a constant current nerve stimulator has been described.[19]

As with continuous infusion of local anesthetics for epidurals, the dose must be regulated, and appears to be less than needed for normal sensory blockade of the arm. Titration and observation are necessary to watch for signs of local anesthetic toxicity as well as changes in the depth of neural blockade (increased motor block and decreased analgesia).

A general rule applies to all forms of infusion techniques (intraspinal narcotics and continuous neural blockade): The patient need never have *any* pain, if these are properly timed. They should be used as prophylaxis against pain, not to treat it after it has occurred. If this is kept in mind, the success rate of these techniques increases significantly.

Interpleural Analgesia. The insertion of a catheter into the space between the parietal and visceral pleura for the infusion of local anesthetic agent is a relatively new technique. It is gaining popularity in some centers for the provision of pain relief following upper abdominal (unilateral) and thoracotomy procedures. Many advantages are claimed for the technique, including improved analgesia and markedly improved postoperative pulmonary function.[21, 25] The data are impressive, and the technique can be performed with a patient supine using an anterior axillary approach.

A needle is inserted in a variant of the "hanging drop" technique using the same landmarks as for insertion of a chest tube. A catheter can be threaded through the Tuohy or Crawford needle 10 cm into the interpleural space. Dilute local anesthetic solution equivalent to 0.125% bupivacaine up to 30 mL/hour can then be infused to provide profound analgesia.

Problems associated with the technique include pneumothorax (up to 6%) and the risk of local anesthetic toxicity.

Interpleural analgesia seems to offer an alternative to epidural block when patient positioning difficulties are likely to occur. It is an easy technique to learn and may offer significant benefit to the properly selected patient. It requires little advance preparation and does offer an alternative for "escalation" of the pain control technique when that is necessary in the PACU.

ASSESSMENT OF RESPONSE TO INTERVENTION

Whatever form of analgesia is used, the patient needs to be evaluated frequently in the PACU. One caveat, however, is that the PACU

Table 5. *Suggested Guide for Chart Review*

LOOK FOR	REASON
Immediate perioperative administration of narcotics	Plan for narcotic loading will be influenced by amount of narcotic patient has received prior to PACU arrival: remember the anesthetic record—total intraoperative narcotic
Longer history of analgesic use preoperatively	A guide to individual dose-response. There may be a suggestion of narcotic tolerance and its severity
Previous response to surgery and postoperative analgesia	A possible guide to successful treatment. A warning about unsuccessful paths
Response to previous use of non-narcotic and nonpharmacologic analgesic techniques	NSAID and acetominophen response a possible guide to successful treatment. Patient's ability to use distraction, relaxation, hypnosis, autohypnosis is a guide
Other problems/sources of nociception	History of coronary artery disease, possible perioperative angina as source of chest pain Obstructive/restrictive pulmonary disease, sources of chest pain Musculoskeletal disease, sources of positioning related pain Metabolic disease, sources of peripheral neuropathic pain Other surgical disease, treated or untreated, additional sources of pain

is a stimulating environment that is not duplicated on the ward. The final half-hour or so of observation should be undertaken with minimal patient interaction, similar to the situation on the ward, to look for the appearance of respiratory depression or excessive somnolence (Table 5).

The use of an acute pain management service is helpful in this respect. They form a team who can continue evaluation of the patient and respond to variances in needs and failures of therapy (Table 6). They should be involved early to make suggestions and to become familiar with the case as soon as possible.

Improvement in Pain Behavior

What Does It Feel Like? The patient should note an improvement in his pain, from whatever source. Analgesia should result in both a diminution of the pain and, very likely, a change in the character of the pain (from sharp to dull).

How Much Pain? The pain should continue to be quantified. Unfortunately, not all analgesic strategies work for all patients; the change in the magnitude of pain may be in the incorrect direction.

Table 6. *Special Pain Management Problems in the PACU*

PROBLEM	DIFFERENTIAL DIAGNOSIS	ACTION
Patient "in pain" and excessively sedated	Emergence phenomenon; residual effects of general anesthesia; residual effects of premedication; other CNS causes (e.g., CVA intraoperatively)	Observe whether pain behavior/sedation is increasing or decreasing; use time to review chart; withhold analgesics
Patient complains of pain in presence of motor/sensory impairment	Residual of receding block	Treat pain; observe regression of deficit
	No block given or impairment not anatomically related	Document; do not treat; consider consultation

This becomes a problem for the patient who was already at "10 on a scale of 10." Do not attempt to achieve "0" as patients are generally satisfied before "no pain" is reached.

How Much Better? The reverse of the pain scale can be used to quantify relief. Rather than "How much does it hurt?" the question becomes "How much relief do you have?" A visual analog scale would have endpoints of "no relief" at the zero point, and "completely relieved" at the other.

This scale is not simply the mirror image of the VAS. The other emotional factors will come into play: relief that analgesia has been achieved; relief that the surgery is over; relief that someone is attending to the pain.

Level of Sedation? Again, the stimulating atmosphere of the PACU may mask excessive sedation. A period of quiet before the final assessment of sedation can be helpful to identify those patients who are excessively sedated. The treatment of oversedation involves observation and time. The use of reversal agents should be avoided if at all possible; if they are used, they should be cleared from the bloodstream before the patient is finally assessed and discharged from PACU.

Level of Motor and Sensory Function? Motor function should be minimally impaired with proper levels of local anesthetics for continuous blockade. Some sensory impairment may be present prior to discharge of the patient, but a block should be shown not to be advancing before patients are discharged to ward care.

SPECIAL CONSIDERATIONS

Long-Range Planning (> 24 Hours)

A comprehensive plan should be established with the acute pain management service, including immediate postoperative care (generally involving parenteral narcotics and/or local anesthetic blocks) for the next 3 to 5 days, and longer range care, including conversion to simpler forms of analgesia. A plan for greater than 24 hours is worth discussing with the surgical team while the patient is in PACU even if there is no acute pain team so that an early approach to treatment that is consistent with the long-range plan can be taken.

Inpatient. What will happen to the patient after he goes to the ward? Have follow-up plans been made? Generally, patients recovering from surgery have 3 to 5 days of severe to moderate pain,[3] which decreases remarkably over that time period until (1) the patient is able to take medications orally, and (2) the amount of narcotic required is low enough to be given orally.

It is not appropriate to expect oral narcotics to be efficacious in the first 24 hours except in relatively minor, soft-tissue surgery that does not violate the pleura or peritoneum. This time period must be covered by the use of parenteral or intraspinal narcotics, possibly with the use of local anesthetic or by formal regional neural blockade.

Ambulatory Patient. The ambulatory patient is frequently problematic. Narcotics must be given so that the patient is comfortable enough to go home, but not to a point of oversedation. The parenteral loading of a drug such as codeine is particularly helpful in this setting. Oxycodone, which is so popular with many surgeons for outpatient procedures, is not available as a parenteral formulation. If oxycodone is to be used, analgesia should be provided in the PACU with intravenous meperidine and the first dose of oral oxycodone given as soon as the patient can take and retain fluids, and at least 45 minutes prior to discharge to home. This allows adequate time to see what side effects will ensue (if any), such as dizziness or nausea, as well as how effective the drug will be overnight. Remember that each combination oxycodone tablet (Tylox, Percocet, Percodan, etc.) contains 5 mg oxycodone, which is about 65% bioavailable and is equipotent to morphine. One oxycodone tablet is thus equivalent to about 3 mg intravenous morphine. Hydrocodone preparations (e.g., Vicodin) are equipotent to oxycodone.

Attempts to hasten discharge by the use of very short-acting narcotics in the ambulatory PACU may well backfire. Very small doses of fentanyl given early in the postoperative period and not followed with a longer-acting drug will likely result in a longer step-down stay while efforts are made to get a patient comfortable enough to travel. A few minutes spent providing appropriate analgesia may actually hasten the patient's return home.

The surgeon must be made aware of the limitations of oral narcotics. On the one hand, the procedure must be limited enough that oral analgesics will be sufficient for postoperative pain relief. On the other hand, there are certain patients for whom even the most limited of procedures will require hospitalization for inadequate analgesia.

There is no absolute contraindication to the use of local anesthetic blocks for the ambulatory patient. A balance must be struck between lack of motor and sensory function and the improved analgesia that these blocks provide.

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Management of Fluid and Electrolyte Problems

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Fluid and electrolyte therapy in the postoperative period is best seen as a continuation of the therapy given before and during anesthesia. Thus, the preoperative condition and the operation itself dictate the outlines of postoperative management. Under ideal circumstances the patient will be normovolemic and the electrolyte and hemoglobin status normal when the patient is transferred to the recovery room; under these circumstances, only maintenance will be needed during the stay in the recovery room. In reality, however, patients coming into the recovery room are often in an unstable condition and need closely monitored fluid therapy. There also may be pre-existing deficits that must be corrected.

An understanding of body composition and of the changes that occur in injury is essential to proper fluid therapy. The abnormal body composition of the injured patient after fluid resuscitation is the result of the therapy superimposed on the body's response to the injury. The daily fluid and electrolyte therapy for such a patient is the sum of normal requirements as well as the additional needs of each specific stage of convalescence after injury.

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BACKGROUND

Body Composition

The adult body is composed of three functional components: fat, cell mass, and extracellular structures. The amount of each varies as a function of age, sex, and body size. Total body water (TBW) is the largest single component of body weight. It is greater in a young muscular adult male (over 60%) than in an elderly obese female (under 45%), because adipose tissue contains little water, and obese individuals and women have proportionally larger amounts of adipose tissue. Age is important because lean body mass decreases with age and thus the ratio of body water to total body weight will also decrease. In general, TBW is considered to be distributed into two major fluid spaces: intracellular fluid (ICF), which makes up 25 to 40% of average body weight, and extracellular fluid (ECF), which makes up 20 to 25% of normal body weight.

The ECF is divided into approximately 11 L of interstitial fluid (ISF) and 3 L of plasma volume, and includes blood, lymph, and transcellular fluid as well as ISF. The ECF is composed of a heterogenous group of fluids and the ion concentration is not exactly the same for each of the spaces. For clinical purposes, however, the Na^+ concentration and ECF will be referred to as a single space. The osmotic and electrical effects, as well as other factors, result in an intracellular Na^+ concentration of 10 mmol/L and an extracellular concentration of 145 mmol/L. Na^+, together with the associated osmotically active anions Cl^- and bicarbonate, are responsible for more than 90% of the solute load of the ECF. As a result, Na^+ and its accompanying anions are the major determinant of the ECF. Cells maintain a low intracellular concentration of Na^+ despite a high concentration in the fluid surrounding them. Magnesium, phosphorus, and potassium are present in relatively low concentrations. Less than 2% of total body K^+ stores are in the ECF and, therefore, K^+ gains do not significantly affect ECF osmolality. The osmality of all body fluid compartments is considered equal because the cell membrane is completely permeable to water. The different ionic composition of ICF and ECF, and osmotic balance, are maintained by selective cell membrane permeability and active, energy-requiring transport of Na^+ ions up the electrochemical gradient. When the osmolality of ECF changes secondary to gain of water or Na^+, however, the redistribution of water between the ICF and ECF compartments is minimized by antidiuretic hormone (ADH) and aldosterone release.

The total exchangeable K^+ can be considered as equivalent to the body stores of K^+ for clinical purposes. A healthy man (70 kg) has a pool of exchangeable K^+ of 2700 to 3400 mEq; a woman, 2100 to

2300 mEq. In contrast to K^+, exchangeable Na^+ is only 65% of total body Na^+. Men contain approximately 2800 mmol of Na^+; women, approximately 2600 mmol. A normal 70-kg man contains approximately 2100 mmol of chloride.

Water Balance

Control of the volume of the ICF and ECF spaces is complex and intimately related to the regulation of solute balance. The supraoptic and paraventricular nuclei of the hypothalamus function as the integrating regions for regulation of water balance, receiving inputs from many areas (Fig. 1). Normally, water intake and output is controlled in response to the osmolality of the ECF. Osmolal regulation is, however, subordinate to the maintenance of ECF volume and, in particular, the intravascular compartment of the ECF volume, This, in turn, is subordinate to perfusion or delivery of essential elements to tissues.

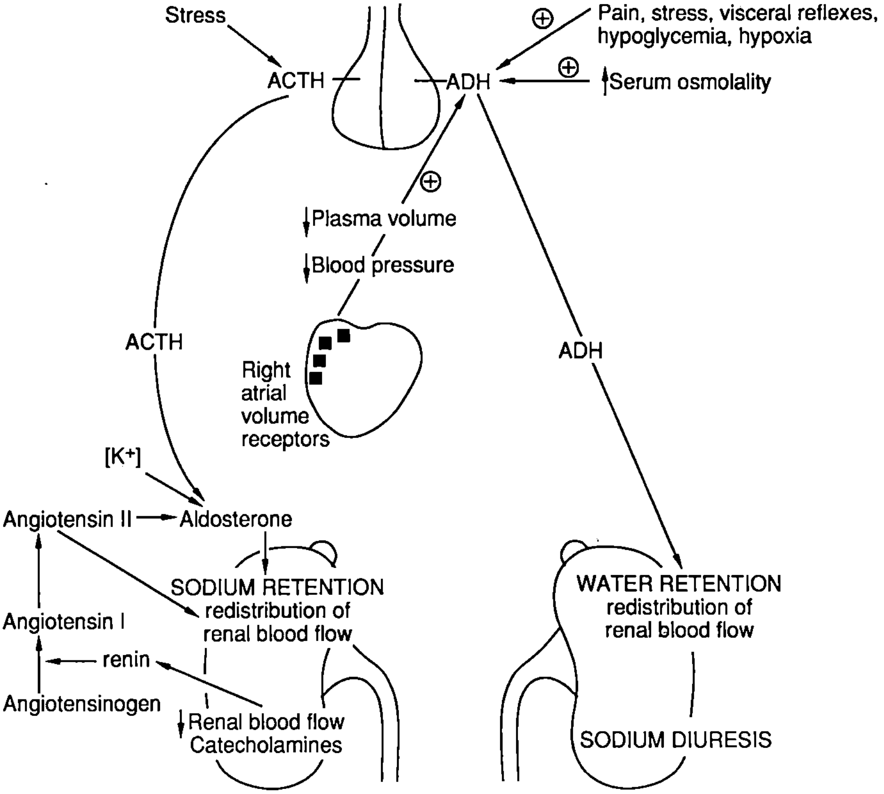

Figure 1. Schematic overview of hormonal control of fluid and electrolyte balance. Adrenocorticotropic hormone (ACTH) is secreted from the anterior pituitary and ADH from the posterior pituitary gland. (*Modified from* Twigley AJ, Hillman KM: The end of the crystalloid era? Anesthesia 40:860, 1985.)

Thus, hypotension stimulates a response by the system even though osmolality and ECF volume appear to be normal.[26]

Water losses take place by three routes: evaporation from the respiratory tract and skin ("insensible losses"), urinary output, and the water content of stool. Insensible losses depend on body size, physical exertion, environmental temperature, and humidity (see section on insensible water loss). Total insensible water loss is between 300 and 500 mL/m of body surface area per day, with minimal activity in a temperate environment. Sweat volume is low at room temperature but may reach several liters a day, with serious losses of both water and NaCl, in warm humid environments with exposure to heating devices and/or when fever is present. Markedly obese patients have an increased water loss, largely due to sweat. Such patients show marked changes in daily weight, consistent with unusually large fluctuations in ECF.

ADH is major factor in controlling urinary output. It is released primarily in response to changes in osmolality, circulating blood volume, and perfusion pressure; lowering of blood pressure increases ADH release (see Fig. 1). In addition, hypoxia and hypoglycemia are capable of stimulating ADH release. Also pain, vomiting, stress, and drugs indirectly affect ADH release. Increases in plasma osmolality of as little as 2% can cause major changes in the rate of ADH secretion. In general, the threshold for ADH release in normovolemic recumbent humans is between 276 and 290 mOsmol.[47]

The two most important effects of ADH are antidiuresis and vasoconstriction. Depending on plasma ADH levels, urine osmolality varies from 50 to 1400 mOsmol. The ability of ADH to develop a highly concentrated urine is enhanced by its action on the renal vessels. ADH redistributes blood flow, with increased inner renal flow and decreased cortical flow, without changing the total flow.[3] In addition, high plasma levels of ADH constrict the renal vasculature, reducing the glomerular filtration rate (GRF), and supplementing the fluid-reabsorbing effect of ADH. Urine output is the difference between the GFR and total fluid reabsorption along the nephron. A reduction in urine output may be the result of reduced filtration or increased absorption. However, even with maximal ADH stimulation, urine outputs usually remain higher than 50 mL/hour if the GFR is normal.[55]

Electrolyte Balance

Electrolytes are discussed in terms of their exchangeable components; minerals that are incorporated into bone, collagen, and connective tissue exchange very slowly with plasma and generally are not considered in determining the composition of parenteral fluid therapy needed to achieve daily balance.

Aldosterone is the major agent altering Na^+ balance (see Fig. 1). Decreased arterial pressures and renal blood flow release renin, which splits angiotensin I from angiotensinogen. Angiotensin I is converted to angiotensin II, which controls the rate of aldosterone secretion. In turn, it is the vasoconstrictor effect of angiotensin II and the Na^+-retaining effect of aldosterone that act to restore the effective circulating volume (see Fig. 1). In addition, a kind of autoregulation seems to exist in the kidney in which the reabsorption of the glomerular filtrate responds to changes in the filtered load of Na^+.[31] Other factors that appear to affect Na^+ excretion are renal hemodynamics, the sympathetic nervous system, "the third factor," prostaglandins, and others.[17] A normal kidney can reduce Na^+ excretion to as low as 1 mmol/day.

K^+ is predominantly (98%) in the intracellular volume (ICV)[16] and therefore plasma concentration is not closely related to the total body K^+. Because of the effects at the level of the cell membrane, however, even small fluctuations in the plasma K^+ level can be clinically significant. A rapid increase in plasma osmolality results in mild elevation in the plasma K^+, apparently because K^+ is drawn out of the cell.[33] K^+ moves in relation to glucose and other substrates of energy metabolism. Insulin decreases plasma K^+ because of the movement of K^+ into liver and skeletal muscle cells. This depends partially on the active transport of glucose. Insulin secretion actually serves as one of the body's first responses to hyperkalemia. Increased plasma K^+ also increases secretion of mineralocorticoids, leading to increased renal K^+ excretion.

Alpha-adrenergic activity results in hyperkalemia, whereas beta-adrenergic activity produces hypokalemia.[6, 60] The infusion of alpha agents such as noradrenaline produces a hyperkalemic effect, whereas administering a beta agonist such as isoproterenol produces a hypokalemic effect. Adrenaline, which is a mixed adrenergic agent, has two effects on K^+ flux: the rapid infusion results primarily in hyperkalemia, whereas a continuous infusion of adrenaline produces hypokalemia.

A definite relationship exists between the serum concentration of Na^+ and K^+ and the total exchangeable Na^+ and K^+; however, there is poor correlation between serum Na^+ and total body Na^+.

$$\text{Serum Na}^+ = \frac{(\text{total exchangeable Na}^+) + (\text{total exchangeable K}^+)}{(\text{total body water})} - 26$$

Thus, serum Na^+ can be increased by the addition of K^+ or Na^+ or by the restriction of free water. Retention of water in excess of Na^+ can lead to hyponatremia even with an excess of total body Na^+. Administration of K^+ can raise the serum Na^+ concentration in hyponatremic patients.

Thus, the absolute reduction of serum K^+ that occurs with depletion can be expected to reflect a very large total body K^+ deficit. A change in acid-base status may alter the serum K^+ levels quite markedly as H^+ is exchanged for K^+. In general, acidosis leads to hyperkalemia, whereas alkalosis leads to hypokalemia, metabolic states having a more pronounced effect on plasma K^+ levels than respiratory states. Alkalosis may lower serum K^+ markedly, particularly if there is a pre-existing total body deficit.

To maintain electroneutrality:

$$\text{serum } (Na^+) + (Ca^{2+}) + (Mg^{2+}) + (K^+) = (Cl^-) + (HCO_3^-) \\ + (\text{protein}) + (\text{sulphate}) + (\text{phosphate}) + (\text{organic acids})$$

$$\text{Thus: } (Na^+) + (K^+) = (HCO_3^-) + (Cl^-) + 14$$

The factor of 14 is commonly termed the anion gap. If (sodium + potassium) − (bicarbonate + chloride) exceeds 18, the substantial addition of an anion must be suspected. These are (1) HPO_4^{2-} and SO_4^{2-}, as in renal failure; (2) lactic acid, as in hypoxia, shock states, and salicylate intoxiation; and (3) keto acids, as in diabetic ketoacidosis. If the anion gap is less than 5, hypoproteinemia is the most likely cause.

EFFECTS OF TRAUMA AND SURGERY ON FLUID AND ELECTROLYTE BALANCE

The induction of anesthesia with a variety of agents has little effect on ADH levels.[9, 44] Surgical procedures, however, are consistently associated with marked elevations in ADH levels. The skin incision causes a dramatic rise in ADH.[9, 44] There is a rough correlation between the magnitude of surgery and ADH levels,[57] but the roles of anesthesia level, blood pressure, osmolality, blood volume, and fluid replacement have not been clarified. ADH levels remain elevated for about 2 days postoperatively.[53]

With increasing severity of injury there is a progressive increase in muscle ECF, Na^+, and Cl^-, while the intracellular space is decreased. Muscle K^+ and magnesium tend to decrease with increasing degree of injury. There seems to be an obligatory expansion of the ECF that is associated with characteristic changes in muscle membrane potential (Table 1). It has been suggested[15] that the postinjury changes in ECF and ICF probably reflect a change in the permeability of cell membranes and a disturbance in ionic exchange. Thus, injury is thought to lead to an increased escape of nondiffusible solutes into

Table 1. *Changes in Membrane Potential and Extracellular Water Following Shock*

	ECF (% BODY WEIGHT)	MEMBRANE POTENTIAL (MV)
Control	13.9	−91
Shock	7.7	−65
Resuscitation	18.4	−90

Modified from Shires GT: Fluid and electrolyte therapy. *In* Inney JM, Egdahl RH, Zuidema GD (eds): Manual of Preoperative Care, ed 2. Philadelphia, Saunders, 1971.

the ECF. This causes a decrease in the osmolality of cell fluids, while increasing the osmolality and resultant volume of the ECF.

Studies using the needle biopsy technique have evaluated the role of nutrition and activity level in muscle fluid sequestration.[4, 14, 36] These studies demonstrated that an increase in muscle Na^+ and Cl^- occurs in noninjured portions of the body. These changes are not affected by the form of nutritional support administered. There was an increase in muscle ECF in these patients, which was unaffected by the nutritional support system administered.

The ECF expansion requires fluid and sodium administration above maintenance levels and results in an increase in body weight. The weight gain after operation or injury is variable and depends on the severity of the injury, preoperative status, and on the perioperative fluid therapy. Elective operations such as total hip replacement or colon resection are commonly associated with a 3 to 5% weight gain, whereas major trauma may be associated with a 10 to 15% gain in body weight. The physiologic effect of this weight gain is variable and depends on the underlying clinical state of the patient. In a young healthy individual, a 10 to 12% weight gain (7 L positive fluid balance) may be well tolerated, whereas a much smaller weight gain may be poorly tolerated by the elderly patient with decreased cardiopulmonary system reserve.

Plasma K^+ levels increase shortly after the onset of trauma with a peak in about 8 hours due to redistribution of K^+ from ICF to ECF, and return to normal over the subsequent 24 to 48 hours. Severe trauma may be accompanied by late hypokalemia, because the excess plasma K^+ is excreted quickly into urine. The net negative K^+ balance is maximal in the first 24 hours and persists for 2 or 3 days. If no complications occur, an average of 60 mmol of K^+ is lost on the day of operation, an average of 35 mmol on the first postoperative day, and about 20 mmol/day for 4 days. Total K^+ loss is 175 mmol, about 5% of the total-body K^+.[50]

CLINICAL CONSIDERATIONS

The requirements for fluids and electrolytes can be divided in three categories:

1. Normal requirements: What does the patient require in water, electrolytes, basic calories, and micronutrients? The calculation of baseline requirements disregards any pre-existing losses, but baseline volumes may require modification in patients with extracellular fluid expansion associated with delusional hyponatremia.
2. Pre-existing deficits or excesses: What deficits (or excesses) does the patient have in water, electrolytes, blood volume, plasma proteins, and micronutrients? What should be done to correct these abnormalities?
3. Abnormal losses: What does the patient require in order to replace ongoing abnormal fluid and electrolyte losses resulting from the disease or its treatment? This includes ECF sequestration. External abnormal loss may be in the form of excessive loss of water and electrolytes by normal routes of excretion or secretion, or losses that may occur from intraluminal tubes, drains, fistulas, or wounds. The most common source of abnormal external loss in surgical patients is the gastrointestinal tract; next in frequency are losses from surgical wounds, increased evaporation from the skin and respiratory tract, and those from direct injury to the skin. The sequestration of extracellular fluids into areas of traumatized or infected tissue decreases ECF without external loss or change in body weight.

A part of the fluid and electrolyte requirement will be obtained through the metabolism of body tissue in a semistarving state. Endogenous water is derived from the shrinkage or breakdown of protoplasm, which is roughly 75% water, as well as the water of oxidation that results from fuel oxidation. This is about 10% of the total daily requirements. However, fever, trauma, and infection will increase endogenous water production along with the increased oxidation of tissue fuel. This water is almost completely sodium free but is rich in potassium, magnesium, phosphate, and sulphate.

The daily caloric requirements of the postoperative patient depend in part on sex and age but much more on the extent and type of surgery, the presence of infection, and the amount of energy required to maintain body temperature in the presence of abnormal evaporative water losses, either from hyperventilation or from evaporative cooling that occurs as the result of extensive thermal injury. The daily caloric requirement of the afebrile patient of average size, at bed rest, will vary from 1300 to 1900 kcal, 400 to 500 kcal of which are often provided by the administration of isotonic glucose into a peripheral vein. The use of glycerol instead of dextrose as an energy substrate after surgery may help to control blood glucose levels in diabetic patients.[30] It can be infused into peripheral lines in low concentrations and its protein-sparing effect in hypocaloric amounts equals that of glucose.

The data listed in Table 2 are intended only as approximate guide-

Table 2. *Baseline Fluid Requirements**

	AGE (YEARS)	FLUID (ML/KG/DAY)
Average adults	25–55	35
Young active adults	16–30	40
Older patients	55–65 (±)	30
Elderly	65	25

* Adult values are based on ideal weight for height and age.

Modified from Randall HT: Fluid, electrolyte and acid base balance. Surg Clin North Am 56:1019, 1976.

lines in fluid replacement. Fluid therapy should be individualized for a given patient. The maintenance requirements can be covered with half-isotonic saline with the addition of 2.5% glucose. This might be the most physiologic solution for this purpose.[54] Fluid administration should be determined by parameters such as blood pressure, central venous pressure (CVP), and urinary output. In general, urinary output serves as a useful guide to fluid therapy and should be maintained at minimum levels of 600 mL/day, or 30 mL/hour.[56] If output decreases below 600 mL/day in association with a decreasing central venous pressure or a decrease in body weight, an increase in fluid requirements is indicated. A decrease in urinary output, associated with a rising CVP and weight gain, may indicate the onset of interstitial pulmonary edema, and diuretic therapy should be started. One should be careful, however, in reading the CVP values.[2] With a low or absent urinary output, before doing anything else, the patency of the urinary drainage system should be checked. It is important to note that from the third to the sixth day following injury, a diuresis will ordinarily develop. If not, there may be impending complications (congestive heart failure, sepsis, renal failure, and so forth).[56]

FACTORS MODIFYING FLUID AND ELECTROLYTE REQUIREMENTS

Gastrointestinal Losses

The normal daily volume of secretion into the gastrointestinal tract has been estimated to be 8000 to 10,000 mL/day, of which saliva constitutes 1 to 2 L; gastric juice, including acid and mucoid secretions, about 500 to 750 mL; and pancreatic juice, approximately 100 mL. Secretion of the upper small-bowel mucosa is between 2000 and 3000 mL. However, all but 100 to 200 mL of the secretions are normally reabsorbed by the small bowel and the colon.

Table 3. *Electrolyte Content of Gastrointestinal Secretions (mmol/L)*

SOURCE OF FLUID	Na^+	K^+	Cl^-	HCO_3^-
Saliva	60	20	16	50
Gastric	30–90	4.0–12	50–155	0
Upper small bowel	70–120	3.0–7.0	70–120	10
Ileum	90–140	3.0–8.0	80–125	15–20
Bile	145	4.0–7.0	80–110	50
Pancreas	120–140	5–8	60–80	

From Randall HT: Fluid, electrolyte and acid base balance. Surg Clin North Am 56:1019, 1976.

Abnormal losses from the gastrointestinal tract include water and electrolytes and varying amounts of protein. Table 3 shows the average content of fluids from different levels of the intestine. It is important to note that, of all the secretions, only bile and pancreatic juice are approximately isotonic in their electrolyte content. The average calculated osmolality of saliva is about 160 mOsmol; of upper small-bowel content, 220 mOsmol; and of fluid from the distal ileum about 240 mOsmol. Other substances, including mucoproteins, other polysaccharides, urea, calcium, and phosphate, add to these approximations of the total osmolality. The values in Table 3 may be used for semiquantitative replacement of gastrointestinal tract losses. When volumes of these losses exceed 2000 mL in 24 hours or when substantial losses continue for more than a few days, it is wise to analyze an aliquot of the 24-hour drainage for electrolytes and protein and to determine the pH of a freshly obtained specimen. More precise replacement can be made with this information. It is important to note that replacement of abnormal losses should be provided in addition to baseline requirements.

Insensible Water Loss

A patient with a fever of 103°F (39.4°C) will require an average of 500 mL of additional water per day. The endogenous water production associated with the hypermetabolism of fever is also increased, but not enough to offset the increased losses. Sweating will increase the average adult water requirements by 500 mL/day for each degree Fahrenheit of ambient temperature above 85°F (29.5°C), depending on the humidity. Sweat is about half normal sodium chloride, so that additional salt must be provided in the therapy. The potassium content of sweat is negligible. The seriously ill febrile patient should be cooled, preferably by air conditioning, because the insensible loss of water from evaporative cooling is very large.

Hypertensive patients receiving chronic diuretic therapy are somewhat vasoconstricted and often salt-depleted. These patients often are dehydrated and require additional fluid. Fluid restriction can result in severe dehydration and possible irreversible renal failure and is contraindicated. These patients may be hypokalemic and should be given potassium with their intravenous fluids.

Mechanical ventilation, if prolonged, has been associated with water retention in about 20% of patients.[52] The addition of positive end-expiratory pressure (PEEP) to mechanical ventilation decreases urine output and free water clearance, whereas urine osmolality increases. Continuous positive pressure breathing probably increases ADH levels by activating volume receptors.[38] The effects of ventilation and PEEP on ADH are not clear and require more studies to separate the effects of blood gas changes, cardiac output, and changes in volume status and pressure. Hyperventilation results in increased water loss by evaporation.

Hyperthyroidism

Hyperthyroidism substantially increases the turnover of water in parallel with increases in caloric requirements. Hyperthyroid patients in the semistarving state tend to consume massive amounts of lean tissue and fat, producing unusual amounts of endogenous water, and simultaneously losing water by respiratory evaporation and skin sublimation as well as sweating.

Type of Anesthesia

There is an association between the type of anesthesia used and the amount of fluid and blood given. With regional anesthesia the area anesthetized and agent used generally will guide fluid requirement. For example, an epidural block for orthopedic surgery necessitates administration of large amounts of fluid. The sympathetic blockade and increased venous capacitance protect against intraoperative fluid overload but with resolution of the anesthetic the affected area may vasoconstrict, which can lead to fluid overload. Therefore, to avoid these postanesthetic problems in certain patient groups the use of vasoconstricting agents (e.g., phenylephrine hydrochloride) should be considered to cut down the large volumes of fluid needed to control blood pressure.

COMMON PROBLEMS

Overhydration

Overhydration can result from overadministration of nonelectrolyte fluids, increased endogenous vasopressin secretion secondary to stress, and renal insufficiency with oliguria. Elderly patients with debility and malnutrition are often mistakenly considered to be dehydrated, and nonelectrolyte solutions are given, resulting in water intoxication. The patient with large volumes of gastric drainage is another candidate for overhydration when gastric aspirate is replaced by an unsuitable solution. Signs of water intoxication include drowsiness, restlessness, muscular weakness, cardiac arrhythmia, fever, convulsions, and coma. Migration of water into the brain tissue from the hypo-osmolar plasma produces cerebral edema. The hematocrit and plasma Na levels are low (see section on hyponatremia). Cessation of fluid administration may reverse mild cases of water intoxication. Advanced cases may require diuretics and hypertonic solutions. Hypertonic solutions reduce cerebral edema and can be given as 50 to 100 mL doses of 3% saline, 50% glucose, or 20% mannitol.

Dehydration

The common use of the term "dehydration" can lead to confusion since there are two different clinical syndromes that may be referred to as dehydration. One syndrome is due to the loss of sodium and chloride with contraction of the extracellular volume, causing decreased skin turgor, a rapid pulse, and a lowered blood pressure. Laboratory findings are related to increased hemoglobin and hematocrit with lower sodium and chloride values in the plasma, although urine volume and concentration are not remarkable. Another syndrome is associated with the primary loss of water without loss of electrolytes. Skin turgor, together with circulatory signs and symptoms, is usually normal even though thirst becomes intense. Oliguria with maximal urine concentration is associated with elevated plasma sodium and chloride. When referring to dehydration, sufficient description should be provided to clarify which of these two syndromes is under discussion. Fever, exposure of the peritoneum during surgical procedures, inhalation of dry gases, and burns all increase evaporative water losses. The accumulation of fluid associated with ascites and pleural effusion, or fluid sequestration in the bowel lumen during intestinal obstruction are other forms of more insidious development of water losses. Patients with ileus usually lose 2 to 4 L of fluid by vomiting and may have accumulated 4 to 6 L in the lumen of the gastrointestinal tract.

A dehydrated patient requires replacement of the losses in addition to daily needs. In general about 50% of the losses should be rapidly replaced over 4 to 8 hours, and the remainder within 16 to 20 hours. Since fluid losses are only roughly estimated, physiologic criteria must be used to warn of overhydration when large fluid volumes are given rapidly. Oliguria, low peripheral temperature, hypotension, and tachycardia indicate the need for more rapid fluid replacement, while CVP values over 15 cm H_2O indicate that fluids are being infused too fast. In mild to moderate dehydration the fluid losses are approximately 3 to 6% of body weight, whereas severe dehydration correlates with approximately 10% fluid loss.

Hyponatremia

The main causes of hyponatremia can be divided into three groups: reduced ECF volume (Na^+ deficit, H_2O deficit), nonedematous ECF volume expansion, and edematous ECF volume expansion. Hyponatremia is frequently observed after surgical and traumatic injury. Increased levels of ADH following injury lead to retention of water in excess of Na^+, dilutional hyponatremia, and expansion of the ECF. This happens during the postinjury phase of maximal Na^+ retention and normalizes when renal Na^+ excretion returns to normal. Cell water released due to catabolism may also contribute to posttraumatic hyponatremia.[37] Excessive use of hypotonic solutions or nonelectrolyte fluids can cause iatrogenic dilutional hyponatremia and the patient has both too much Na^+ and too much water. The treatment of choice is fluid and Na^+ restriction with or without diuretics. Hyponatremia and hypotonicity are common also in starvation, malnutrition, and other depleted states. In the latter situations, failure or inadequacy of the Na^+ pump due to depletion of energy stores probably leads to the accumulation of intracellular Na^+ and water.[50] Replenishment of energy stores as well as excretion of excess water is needed to reverse the condition.

Hypernatremia

Marked dehydration and desalting water loss are usual reasons for hypernatremia. Loss of hypotonic body fluids, such as gastric juice, results in ECF depletion and hypernatremia. Paradoxically, an elevated serum Na^+ usually means a depleted total body Na^+ with an ECF deficit. Hypovolemic hypernatremia may also result from diuretic administration and glucosuria and inappropriate replacement of evaporative loss from skin and lungs. The treatment is repletion of the ECF space. The electrolyte concentration must be calculated on the basis of fluid lost. If these lost fluids are replaced with hypertonic Na^+ solution, hypernatremia will persist.

Hypokalemia

Hypokalemia may be defined as plasma K^+ concentrations less than 3.5 mmol/L. However, normal plasma K^+ values can be associated with intracellular and total-body K^+ depletion. A K^+ deficiency can be due to reduced intake during malnutrition and starvation as well as chronic losses due to wasting diseases, severe diabetes, sepsis, carcinomatosis, prolonged complicated postoperative states, multiple organ failure, diarrhea, chronic use of diuretics, and prolonged vomiting. Hyperfunction of the adrenal cortex (Cushing's disease) or overadministration of steroids also deplete total body K^+. Hypothermia may cause transient hypokalemia.[5]

With severe vomiting and prolonged nasogastric suction, a loss of both H^+ and K^+ occurs, leading to a hypokalemic alkalosis; the hypokalemia makes the alkalosis worse and alkalosis makes the hypokalemia worse. Hyperventilation further worsens the situation.[1] Usually, however, chronic respiratory acid-base disturbances result in minimal disturbances of S-K^+. This combination may be particularly hazardous, since the loss of plasma volume associated with vomiting stimulates aldosterone secretion and the adrenal stress response, which aggravate the hypokalemia. Ordinarily, the loss of H^+ by vomiting would be compensated by the loss of OH^- in the urine, resulting in an alkaline urine. However, the aldosterone response initiated by the hypovolemia leads to resorption of Na^+ and HCO_3^- by the kidneys with increased urinary K^+. This paradoxical aciduria tends to aggravate the alkalosis, which in turn further worsens the hypokalemia.

In postoperative patients, hypokalemia can be a result of chronic changes in K^+ balance or be the result of sudden alterations. In respect to cardiac complications, the acute changes seem to have greater importance.[62] Chronic moderate hypokalemia should not cause too much concern in the otherwise uncomplicated recovery room patient.[58, 62] However, in some situations, dangerous hypokalemia may develop. Acute hypokalemia, due to either pathologic losses or transmembrane shift of potassium into the cell, may produce significant cardiovascular problems because there has not been sufficient time for physiologic compensatory mechanisms to act effectively.[62] In otherwise healthy patients, S-K^+ of less than 2.5 mEq/L is associated with a real danger of cardiovascular problems; supraventricular tachyarrhythmias may occur even with S-K^+ around 3.0.[35, 62] Pre-existing cardiac problems, and medication (e.g., digitalis), poor medical condition, and extensive surgery greatly increase the risks of hypokalemia.[35]

It seems reasonable to aim at normalization of any hypokalemia found in the recovery room. However, overzealous correction should be avoided as it in itself is potentially life-threatening. Intravenous infusion of K^+ should normally not exceed 20 to 30 mEq/hour (0.5 mEq/kg). Usually, an infusion rate of 5 to 10 mEq/hour, i.e., 120 to 240 mEq/day, is adequate. If rapid infusions are thought to be required, the cardiac monitoring should be meticulous. The goal of rapid intravenous infusion is to fill the small extracellular K^+ space. It is important to recognize that intravenously administered K^+ may take 8 to 48 hours to be incorporated intracellularly.[41] Hypokalemia, alkalosis, and dehydration must all be corrected. It is necessary to begin correction of the hypokalemia before attempting to correct dehydration by massive saline infusions because the dilution will further lower plasma K^+ levels and may precipitate arrhythmias or convulsions. In hypokalemic patients, hypomagnesemia may be present and should also be corrected.[62]

Hyperkalemia

Hyperkalemia in the recovery room is mainly due to renal failure or iatrogenic problems, including rapid administration of K^+ and massive transfusions of stored blood. It may also result from large tissue damage like multiple trauma and severe burns. Hyperkalemia is usually associated with hyponatremia and hypocalcemia. Poor perfusion and metabolic acidosis also result in hyperkalemia. The manifestations are mainly symptoms of the cardiovascular system and gastrointestinal tract. Nause, vomiting, and diarrhea are common. A steep rise in K^+ levels to 10 mEq/L or more, as is seen with the use of succinylcholine in burned patients, may result in fatal arrhythmias. Acute hypercapnia may also induce a dangerous increase in S-K^+.[62]

Treatment in less severe cases consists of hypertonic glucose solutions, insulin, diuretics, intravenous fluids, and correction of the hypocalcemia and acidosis. The acidosis can be corrected by sodium bicarbonate infusions. Infusion of a glucose/insulin mixture will increase the intracellular shift of potassium and significantly reduce S-K^+. Intravenous calcium gluconate and calcium chloride have also been used to combat the cardiac arrhythmias. Intravenous β_2 stimulators can decrease S-K^+. If S-K^+ exceeds 6 mEq/L, peritoneal or hemodialysis should be considered.

TURP Syndrome

Transurethral resection of the prostate (TURP) carries unique fluid and electrolyte complications because of the need to use large volumes of irrigating fluid for the endoscopic resection. The TURP syndrome is a vascular overload caused by the rapid absorption of irrigating fluid. Near to isotonic 1.5% glycine solution is currently the most frequently used irrigation fluid, but systemic absorption of the irrigating solution can still produce serious complications like hypervolemia, electrolyte dilution (hyponatremia), hypoproteinemia, and toxic levels of glycin.[8, 19–22, 42] The absorption of irrigating fluid takes place both directly into severed prostatic veins and extravascularly. The amount of absorption depends on three main factors: the surgical technique, duration of exposure, and the hydrostatic pressure of the irrigating fluid.[42] Rapid absorption through venous sinuses produce immediate dilutive changes causing significant falls in serum Na^+ during and after TURP.[21] The extravasation of irrigant fluid elicits only mild changes in serum Na^+, and these changes take longer to evolve.[21, 23] Absorption of more than 1000 mL of irrigating solution[23]

Table 4. *Symptoms Associated with the Absorption of Irrigating Fluid in Patients Undergoing Transurethral Prostatic Resection (TURP Syndrome)*

Mild degree (S-Na$^+$ >125 mEq/L)	Paresthesia, nausea, headache
Severe degree	Hypotension, circulatory collapse, hypertension, angina pectoris, ST depression on the ECG, dyspnea, pulmonary edema, transient blindness, confusion, coma

may cause a variety of symptoms during and after surgery (Table 4) depending on the amount of fluid absorbed.[8, 20, 21, 23, 42] Absorption of 3 to 4 L of irrigating fluid seems to be necessary to produce severe TURP reactions, and the associated fall in S-Na$^+$ is between 20 and 50 mmol/L.[20, 21, 35, 45] The combination of hypervolemia, hyponatremia, and hypoproteinemia promote the efflux of intravascular water into the other compartments, and signs of pulmonary and cerebral edema may occur. Cardiovascular collapse and death have been reported after massive absorption of standard 1.5% glycine irrigating solution.[8] Although the symptoms often first present intraoperatively, they may start or get worse after transfer to the recovery room.[6, 8, 23]

When no central nervous system symptoms are present and S-Na$^+$ is higher than 125 mEq/L, restriction of hypotonic fluid intake in the recovery room is sufficient.[7] In severe cases more aggressive therapy is needed. To restore isotonicity, hypertonic Na$^+$ infusions (3 to 5% saline, 8.4% sodium bicarbonate) have been used.[7, 8, 42] The use of hypertonic solutions carries two dangers: the volume expansion combined with the already existing hypervolemia may cause cardiac failure; second, a too rapid correction of severe hyponatremia may cause brain damage.[7] Both loop diuretics (i.e., furosemid, 40 mg) and hypertonic solutions can be used to treat fluid overload.[8, 42] These patients must be closely monitored, and the goal should not be immediate normalization of S-Na$^+$, but avoidance of severe brain edema and cardiopulmonary collapse.

To facilitate early detection of the TURP syndrome, the irrigating fluid can also be marked with low-concentration ethanol. Absorption is immediately detected from measurement of the ethanol content of expired breath, using a portable breath alcohol analyzer.[23] Using 1% ethanol eliminates the euphoria and dizziness occasionally seen with 2% ethanol, and the sensitivity of the test is adequate for detecting absorption.[23]

SELECTING THE PLASMA REPLACEMENT FLUID

In the face of massive blood loss the need for replacing red cells is obvious, but the choice of the asanguinous fluid for resuscitation raises disagreement.[27–29, 32, 39, 50, 51, 56] The "colloid versus crystalloid" controversy is to some degree artificial and centers on philosophy, economics, and side effects. The proponents of the use of colloids (albumin, dextran, modified gelatin, hydroxyethyl starch) as part of fluid therapy argue that resuscitation with crystalloid alone dilutes plasma proteins, thereby reducing plasma oncotic pressure. Reduced oncotic pressure favors fluid movement from the intravascular to the interstitial compartment and thereby predisposes the patient to the development of interstitial pulmonary edema. Also, resuscitation with colloids is more rapid than with crystalloids. All artificial colloids carry a small risk of allergic or pseudoallergic reactions.[46] Large quantities of colloids have also been associated with a greater dependency on ventilator support and a detrimental effect on renal function.[27, 32] Albumin infusions are generally confined to patients whose serum albumin is below 2.5% and who are also receiving nutritional repletion. In the absence of infection, the capillary bed is considered to be intact; therefore, a rise in serum albumin level may be expected with albumin infusion.

The use of hypertonic saline has received much attention. It may be preferable to 0.9% saline in situations in which excess free water administration is not desired and in which interstitial edema is detrimental to function and survival.[11, 48] If hypertonic saline has been used preoperatively or during the operation, it should be remembered that although the hypernatremia and hyperosmolality usually are resolved rapidly, large amounts of potassium may be needed to compensate for increased kaliuresis. The serum potassium level should be monitored closely and replaced aggressively.

SUMMARY

A variety of factors dictate fluid and electrolyte therapy in the postoperative period. Fundamental to provision of therapy is a knowledge of body composition. Although sodium is the major extracellular ion, it is important to recognize that potassium is the major intracellular ion and to a degree, more prevalent on a whole body basis than sodium. The distribution of total body water between the two major fluid spaces (intracellular fluid as well as extracellular fluid) change during stress conditions, and fluid resuscitation therapy requires a clear-cut understanding of the changes that occur. A common problem in the postoperative patient is control of serum sodium. In this regard it is important to recognize that serum sodium is related to both total body sodium as well as total body potassium divided by total body water. Thus, changes in potassium will result in changes of serum sodium. Factors that modify fluid and electrolyte requirements include gastrointestinal losses, insensible water loss, pre-existing deficits due to chronic diarrhetic therapy, mechanical ventilation, hyperthyroidism, and the type of anesthetic technique employed.

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Ventilatory and Pulmonary Problem Management

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Postoperative respiratory management in the postanesthesia care unit (PACU) includes extubation and weaning from mechanical ventilation, management of the ventilator, and monitoring, prevention, and treatment of postoperative pulmonary complications. This article offers some insight into the rationale for and treatment of postoperative pulmonary complications and the routine use of mechanical ventilation.

EXTUBATION AND WEANING FROM MECHANICAL VENTILATION

In routine circumstances, the patient, once he emerges from anesthesia with a normal arterial blood gas on the ventilator, can be empirically discontinued from the ventilator and extubated. The patient can then be placed on 30% to 50% oxygen via an aerosolized face mask and monitored with a subsequent arterial blood gas in 20 to 30 minutes to document stability.

A more conservative approach is to place the patient on successively decreasing respiratory rates on synchronized intermittent mandatory ventilation (SIMV) mode until the patient is on an SIMV of 2 or 4 with a good arterial blood gas and extubate.

Another approach is to place the patient who is on an SIMV or assist-control mode (AC) of 8 to 15 respiratory rate to a T-piece for 30 minutes at a fraction of inspired oxygen that exceeds by 10% what the patient was receiving on mechanical ventilation. If the patient is com-

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Table 1. *Pulmonary Function for Successful Weaning from Mechanical Ventilation*

Oxygenation	
Pa_{O_2}/Fi_{O_2}	>200 mm Hg
$P(A\text{-}a)O_2(Fi_{O_2} = 1.0)$	<350 mm Hg
Fi_{O_2}	<45%
Ventilation—mechanics	
Tidal volume	>5 mL/kg
Vital capacity	>10 mL/kg
Respiratory rate	<20 breaths per minute
Ventilation—endurance	
Minute ventilation	<10 L/minute
Muscle strength	
Maximum inspiratory pressure	< −25 cm H_2O

fortable with a good arterial blood gas he can be extubated and placed on an aerosolized face mask. Although there is controversy about the best method of weaning from mechanical ventilation,[81] a recent prospective trial comparing IMV versus T-piece weaning in 165 medical and surgical patients found no difference between groups.[140] Average weaning time was 5.3 hours in the IMV group and 5.9 hours in the T-piece group. Both groups complied with the criterion for weaning.

When the clinician is uncomfortable and wants to extubate a patient with specific guidelines, objective weaning criteria can be used[113] (Table 1). In general, a patient with a low respiratory rate and fraction of inspired oxygen under 45% with a negative inspiratory force less than −25 cm H_2O will do well during weaning.

Criteria implying a lack of success during a weaning trial can be seen in Table 2.[130] If these problems are not present during the end of a weaning trial, i.e., at 30 minutes, an arterial blood gas can be drawn and the patient extubated if the gas is acceptable. Any patient who is unable to be weaned from mechanical ventilation approaching 12 to 18 hours should be considered for transfer to an intensive care unit for management.

Table 2. *Indicators of Unsuccessful Weaning from Mechanical Ventilation*

Heart rate increased or decreased by >20 beats per minute
Respiratory rate increased by >10 breaths per minute
Diastolic blood pressure increased or decreased by >20 mm Hg
Decreased level of consciousness
Development of cardiac arrhythmia
Evidence of respiratory muscle fatigue (paradoxical breathing)
Anxiety unrelieved by reassurance
Desaturation below 90% by pulse oximetry

From Stone AM, Bone RC: Successful weaning from mechanical ventilation. Postgrad Med 86:315–319, 1989; with permission.

Trials of early extubation within 5 hours versus prophylactic mechanical ventilation for longer than 15 hours have been evaluated in patients after abdominal surgery[115] and coronary artery bypass operations.[102] It was thought that this type of patient with abdominal or thoracic surgery, owing to an element of pulmonary compromise, might benefit from more prolonged ventilation to minimize pulmonary complications. However, there was no difference in the incidence of pulmonary or cardiopulmonary complications in patients undergoing prolonged mechanical support versus rapid extubation. All patients undergoing prolonged ventilatory support required more sedation and pain relief with narcotics to maintain comfort on the ventilator compared with patients undergoing early extubation. Therefore, the earliest possible extubation from mechanical ventilation is recommended after operation.

Monitoring in the PACU should include noninvasive oximetry, which may reduce the incidence of anesthesia complications.[31] One particularly good single-blind prospective study of pediatric surgical patients found that only 10 of 24 episodes of major hypoxemia were detected by the anesthesia team.[34] All 24 events were associated with a saturation below 73%. The total time of hypoxemia was also greater in the group without oximetry information; 42 minutes in the group without information available versus 27 minutes in the group with immediate access to oximetry readings.

MANAGEMENT OF VENTILATION

Modes of Ventilation

Mechanical ventilatory modes that are often used in the postoperative period are controlled mechanical ventilation (CMV), intermittent mandatory ventilation (IMV), and assist-control ventilation (AC). The CMV mode provides ventilation to the patient at a given operator-selected rate and tidal volume that cannot be increased or decreased by patient effort. This mode should only be used in the comatose or heavily sedated patient because its use will result in a frustrated patient who cannot breath spontaneously despite the attempt. Because of this, CMV has fallen out of favor and either AC or IMV is used.

AC allows the patient to breath spontaneously at his own rate. The patient's inspiratory effort lowers the pressure in the respiratory circuit and triggers the machine to deliver a preset tidal volume. A minimum fail-safe rate of 8 to 12 is dialed in for the patient who is not spontaneously breathing. This allows a smoother transition to the spontaneous breathing state during emergence from anesthesia and is more comfortable to the patient. Disadvantages of the method include an increased mean intrathoracic pressure with hypotension, particularly if the patient is hypovolemic, and respiratory alkalosis when an increased respiratory drive is present. Fluid resuscitation and change to an IMV setting will correct each problem respectively. AC may be the best mode for acute respiratory distress syndrome, which tends to have high minute ventilation requirements and when the minute ventilation is changing rapidly. With AC the delivered minute ventilation can be rapidly increased with minimal patient effort. With IMV the work of breathing could become too intense with a sudden increase in minute ventilation in a borderline patient with subsequent CO_2 retention.

IMV or SIMV is the other popular mode of therapy used in the postoperative state. This mode delivers a preset respiratory rate and tidal volume with each breath synchronized or designed to deliver a breath that does not interfere with the patient's own spontaneous tidal volume if present. The minimum starting rate should be 8 to 12, with adjustments based on arterial blood gas analysis. The patient is free to take spontaneous tidal volumes between machine breaths by inspiring and opening up the demand valve on the machine, which takes some effort. The size of the spontaneous tidal volume varies with lung compliance, respiratory effort, the diameter and length of the endotracheal tube, compliance of the respirator tubing, and the characteristics of the demand valve on the respirator used. Assuming a large diameter endotracheal tube (8 Fr or greater), a ventilator with a sensitive demand valve, and high compliance respirator tubing, this mode of therapy should work well. Because spontaneous breathing is associated with lower mean intrathoracic pressures, this type of mode is theoretically better when cardiac output is marginal. Because most patients undergoing postoperative mechanical ventilation have normal heart and lungs, the type of ventilator and mode should not be of critical importance.

Tidal Volume and Positive End Expiratory Pressure

The tidal volume in mechanically ventilated patients has traditionally been recommended to be between 10 to 15 mL/kg instead of the normal physiologic breath of 5 to 7 mL/kg. Tidal volume ventilation with 5 mL/kg has been shown to lead to progressive hypoxemia in anesthetized subjects,[45] presumably due to progressive microatelectasis. Lung compliance studies reveal the best lung compliance at 10 to 15 mL/kg with no improvement in compliance or oxygenation above 15 mL/kg.[132] No improvement in alveolar arterial oxygen gradient was noted above 15 mL/kg.[79] In patients with loss of lung volume after pulmonary resection, thoracoplasty for tuberculosis, and other restrictive diseases, reduced ventilator volumes need to be used. One guide is to ventilate the patient at a number of different tidal volumes and calculate total static compliance. The tidal volume with the highest compliance is then selected as the delivered tidal volume.[18]

One recent study has now questioned whether lower tidal volumes (6 mL/kg) with the addition of positive end expiratory pressure (PEEP) might not be just as sensible as supraphysiologic tidal volumes. [78, 101] The added PEEP will prevent the hypoxemia that could occur with previous low tidal volume breathing. There were fewer pulmonary infections in the low tidal volume group with lower mean and peak airway pressures, which could translate into less barotrauma over the long-term. Further studies need to be done on this approach to the optimum tidal volume.

PEEP and continuous positive airway pressure (CPAP) on the ventilator are modes that increases functional residual capacity in ventilated patients and can improve oxygenation. Often CPAP is set at 5 or less because this approximates or is slightly above physiologic PEEP (epiglottic). Zero end-expiratory pressure (ZEEP) is also acceptable if the arterial blood gases are favorable and is preferable in patients with obstructive lung disease.

RESPIRATORY PHYSIOLOGY AND PULMONARY FUNCTION

Respiration and Respiratory Failure

Respiration is defined as the passage of gas molecules across permeable membranes.[95] The two clinically significant gases crossing the alveolar membrane are carbon dioxide and oxygen. Therefore, the inability to maintain an adequate Pa_{O_2} or appropriate Pa_{CO_2} level in the blood is respiratory failure (RF), assuming the person is not at high altitude and has normal cardiac function. Respiratory failure can be further divided into three types.[59] Type I is hypoxemic RF characterized by a low Pa_{O_2} of oxygen and normal or low Pa_{CO_2}, caused by collapse or filling of alveolar air spaces. Type II is hypoxemic–hypercarbic RF or ventilatory failure. Type III RF is characterized by a low Pa_{O_2} associated with and partly caused by an elevated Pa_{CO_2}. The etiology of the elevation in carbon dioxide is due to alveolar hypoventilation and ventilation perfusion mismatch. Type III RF, or perioperative RF,[150] is a syndrome that is not often mentioned as a separate clinical type because of its multifactorial causes and is generally seen in the elderly patient, previous smoker, or obese patient who is in the postoperative state.

Part of the etiology for type III RF is related to the physiology of breathing and changes in lung volumes with age, obesity, and smoking. Normally at end expiration the remaining lung volume not used for expiration is called the functional residual capacity (FRC). It is composed of about 50% of total lung capacity and is divided approximately equally into expiratory reserve volume (ERV) and residual volume (RV). The RV, lung volume at the end of a maximally forced expiration, essentially represents gas volume trapped behind closed airways in which the pleural pressure exceeds intraluminal pressure when people are in their teens, and is present in gravity-dependent regions. The lung volume at which dependent airways tend to close increases with age, smoking, and fluid overload, owing to decreased elastic recoil of the airways. This volume, called closing capacity, becomes greater than residual volume with age alone and becomes greater than functional residual capacity by the age of 65.[76] This erosion into normal tidal volume breathing causes right-to-left shunting and exists at rest in the elderly and creates a relative hypoxemia. Functional residual capacity also decreases in the supine posture or with other causes of increased intra-abdominal pressure such as obesity, abdominal surgery, or ascites. These abnormalities, when combined with postoperative pulmonary dysfunction, neuromuscular weakness due to age, poor nutritional state, and medical illness, predispose to mechanical respiratory support on a ventilator, often for a prolonged period of time.

Postoperative Pulmonary Function

Pulmonary function tests taken soon after upper abdominal surgery show a restrictive deficit that lasts longer than 1 week.[1, 35, 75, 87] There is a 60% drop in vital capacity (VC) and forced expiratory volume in 1 second (FEV1) with no change in the FEV1/FVC ratio on postoperative day 1, which improves to a 30% drop at 7 days. Post-thoracotomy operations not requiring pulmonary resection have a 40% drop in VC on day 1 with recovery by day 7.[1] Surgery of the lower abdominal cavity results in a 35% drop in VC on day 1 with recovery by day 7. There is no change in pulmonary function in patients having surgery outside of the abdominal and chest cavities.

Tidal volume (TV) decreases by about 25% and respiratory frequency increases on day 1 after upper abdominal surgery with no significant change in minute ventilation.[1, 149] By day 7 the TV is normal with a persistent elevation in respiratory rate. There is a drop in TV of 25% on day 1 after lower abdominal surgery with no significant change in respiratory rate. The TV returns to normal on day 2.

The FRC exhibits no drop immediately after operation and a subsequent 30% drop at 16 to 24 hours after upper abdominal surgery.[1] The FRC recovers to normal at about 7 to 10 days. There is no significant drop in FRC with thoracic surgery that does not require pulmonary resection,[1] and lower abdominal and extraperitoneal surgery.

Diaphragm activity is reduced in the immediate postoperative period after upper abdominal surgery, with a shift from predominantly abdominal to rib cage breathing.[52] The reduced diaphragm excursions during tidal and deep breathing are not suppressed by postoperative pain relief,[121] and the dysfunction appears unrelated to anesthesia.[108] The latter effect was shown by studying three groups of mongrel dogs under general anesthesia. Six dogs received no surgery (control), nine dogs underwent upper abdominal surgery (cholecystectomy), and six dogs underwent lower abdominal surgery (pseudoappendectomy). Diaphragm function was assessed by changes in transdiaphragmatic pressure swings, the ratio of changes in gastric to esophageal pressure swings, and the ratio of changes in abdominal to rib cage diameters during quiet tidal breathing. In the upper abdominal surgery group there were significant postoperative decreases in all parameter of diaphragm function. In the control and lower abdominal surgery groups there were no significant postoperative changes. This implies that anesthesia and lower abdominal surgery do not effect diaphragm function. The mechanism of the surgery-induced dysfunction is unknown.

POSTOPERATIVE RESPIRATORY COMPLICATIONS

The four major respiratory complications encountered in the PACU are hypoxemia, hypercarbia, airway obstruction, and aspiration pneumonia. The first two, defined by blood gas abnormalities, are usually mild and usually correctable. These two abnormalities can, however, because of underlying disease processes, progress to respiratory failure.

Hypoxemia

The mechanisms causing hypoxemia are sixfold: ventilation/perfusion (V/Q) mismatch, right-to-left intrapulmonary shunt, alveolar hypoventilation, diffusion abnormality, decreased fraction of inspired oxygen, and decreased mixed venous oxygen partial pressure.[146]

Hypoxemia in the postoperative period can have two temporal patterns. After minor operations in patients without pre-existing lung disease, arterial hypoxemia with or without hypercarbia is often present in the PACU in the first 2 hours.[82] Causes of the early postoperative hypoxemia are related primarily to general anesthesia. They include respiratory depression with alveolar hypoventilation from mild narcotic or anesthetic overdose; loss of pulmonary vasoconstriction reflexes after general anesthetics[83]; increased oxygen consumption due to hypertonic muscles and shivering; depressed cardiac output; increased venous admixture; and V/Q mismatch. Dilution hypoxemia can occur during the first 10 minutes after anesthesia with nitrous oxide if the patient is given a low or room air inspired oxygen fraction.[51] This is due to re-equilibration of nitrous oxide in the bloodstream into the alveolus after the nitrous oxide is discontinued. This dilutes the inspired oxygen and nitrogen, causing a lower concentration of both gases, which can cause a transient hypoxemia. If nitrogen was a more soluble gas (nitrous oxide is 31 times more soluble than nitrogen) in blood than nitrous oxide, this would not occur due to rapid equilibration of nitrogen in the bloodstream and maintenance of a reasonable inspired oxygen fraction in the immediate postoperative period.

A second pattern of gas exchange abnormalities associated with hypoxemia at greater than 2 hours, and the most common cause of postoperative hypoxemia is atelectasis. It is associated with surgery that affects the ability of the patient to breath deeply or causes immobilization in bed. This defect is usually seen in surgery of the upper abdomen or thorax and resolves over a period of 2 weeks.[74] The mechanisms for the hypoxemia are V/Q mismatch secondary to disordered ventilation related to aging,[71] the surgery,[54] and shunting.[120] Another overlapping mechanism that causes part of the mismatching of ventilation with perfusion is related to the relationship between closing capacity and FRC, as mentioned previously.[76]

One type of atelectasis, called microatelectasis or miliary atelectasis, involves no detectable abnormalities on chest roentgenogram or by clinical examination, but arterial blood gas analysis reveals an increase in alveolar arterial oxygen gradient and relative arterial hypoxemia.[60, 97, 119] It is most likely associated with a reduction in pulmonary compliance.[49] A more restrictive clinical definition of atelectasis, sometimes called macroatelectasis, consists of rales, fever, sputum production, and radiographic alterations.[55, 114] Using this latter definition, atelectasis occurs in 20% to 30% of patients after upper abdominal surgery, 5% of patients after lower abdominal surgery, and 1% of patients undergoing surgery outside the pleural and peritoneal cavity.[70, 97, 100] When cardiac surgery is considered, the incidence of atelectasis in the postoperative period is the highest at 91%.[24]

Risk factors, other than surgery, that increase the incidence of atelectasis include the presence of obstruction on pulmonary function tests,[129] smoking 10 or more cigarettes a day,[92] even with normal pulmonary function,[26] obesity,[75, 88, 131, 141] and anesthesia times longer than 3 to 4 hours.[42, 88] One recent study, however, found no increased incidence with different anesthesia times.[110] Sex is no longer thought to be a risk factor when similar smoking histories are taken into account,[148] and the type of anesthesia has little bearing on the rate of pulmonary complications.[33, 42, 70] Age as a risk factor is controversial, with some studies revealing an increased risk[110, 137] and other studies noting no detectable risk.[75, 148]

Prevention of postoperative atelectasis and often subsequent pneumonia includes determining preoperatively which patients are at higher risk. Although preoperative pulmonary function tests are advocated by some,[129] the majority believe a good history and physical examination are as good in identifying the high-risk pulmonary patient.[21, 25, 114] A history that includes smoking, presence of recurrent pulmonary infections, occupational background, asthma, exertion necessary to bring on dyspnea, and presence of chronic bronchitis will alert the physician. If there is doubt or a significant history of pulmonary disease, a baseline chest roentgenogram and arterial blood gas should be obtained with or without preoperative pulmonary functions studies.

Many different methods have been used to prevent postoperative atelectasis. The techniques most widely used are geared toward restoring the restrictive pulmonary deficit with its accompanying increased work of breathing and hypoxemia. The etiology of the restriction is related to small tidal volumes with no sigh breaths, which over a period of hours results in atelectasis, which may be difficult to reverse. To minimize this phenomenon the sigh can be artificially introduced. This is done by encouraging the patient to inspire deeply. The deep breath will produce an increase in transpulmonary pressure and distend the lungs. Studies in humans have indicated that alveoli, once inflated, remain inflated for at least 1 hour.[50, 85] Therefore, any respiratory maneuver should be carried out hourly to maintain sustained alveolar inflation and maintenance of normal lung volumes. An additional maneuver, sitting in a chair, has been shown by Meyers and associates[87] to promote an increase in FRC compared with recumbent beta rest in patients who had undergone upper abdominal surgery.

The most popular method used to improve postoperative pulmonary function appears to be incentive spirometry (IS) used in 95% of hospitals, followed by chest physical therapy in 83%, intermittent positive pressure breathing (IPPB) in 82%, continuous positive airway pressure (CPAP) in 25%, and blow bottles in 17%.[94] Randomized studies indicate that deep breathing exercises with[11, 36, 64] or without[137] an adjunctive breathing device (IS) are superior to no treatment,[137] conventional chest physical therapy,[11, 36] and IPPB.[64, 142] One study looking only at conventional chest physiotherapy with deep breathing versus IS in 103 laparotomy patients revealed a 2% incidence of pulmonary complications with deep breathing exercises alone and a 5% incidence with IS. The general conclusion from these studies is that deep breathing exercises of any type decrease pulmonary complications.[10] The most popular mode of deep breathing is with a quantifiable IS device. No specific IS device appears to produce better results than any other.[77] It is recommended that all patients, particularly high-risk chronic obstructive lung disease patients undergoing upper abdominal or chest surgery, have preoperative instruction in deep breathing exercises prior to surgery. The ideal deep breath should be one that is held for 3 seconds. A 3-second deep breath was found to be superior to multiple deep breaths or a single unsustained deep breath.[144] The deep breathing maneuver used should be supervised hourly by a nurse or respiratory therapy practitioner because preoperative patient education and training alone are not effective in decreasing postoperative pulmonary complications.[107]

Continuous positive pressure mask systems that cover both the nose and mouth have decreased the incidence of postoperative pulmonary complications compared with no treatment[3] or IS.[105, 127] The only disadvantages are the expense and complexity of the system, patient discomfort from a tight-fitting mask, and the potential for aspiration of gastric contents.[126] A more feasible system in the future may be nasal CPAP.[43] In the four cases reported so far, this system was able to clear up atelectasis resistant to chest PT, IS, and bronchoscopy.

In conclusion, the prevention of postoperative atelectasis and hypoxemia is only partially successful at this time in patients with upper abdominal and thoracic procedures. The routine measures to minimize postoperative atelectasis should include the following:

1. Preoperative screening of high-risk patients by history, physical examination, and possibly chest roentgenogram, arterial blood gas, and so forth.
2. Preoperative training of high-risk patients in the use of deep breathing exercises of some type.
3. Breathing exercises that emphasize a large inspiratory effort of at least 3-seconds duration.
4. Supervision of breathing exercises at least once per hour while awake.
5. Adequate pain relief to allow deep breathing.[139]
6. Encouragement, when possible, to be in an upright position in bed.
7. Sitting upright in a chair as soon as possible.
8. A trial of mask CPAP or nasal CPAP for refractory atelectasis.

Pulmonary edema in the postoperative period can cause hypoxemia. The most likely types of pulmonary edema in this setting are due to elevated hydrostatic pressure,[32] decreased interstitial fluid pressure,[65, 145] and increased capillary permeability edema due to a multitude of causes including sepsis. The treatment for all types is the same. Treat the underlying cause and maintain adequate oxygenation with face masks or mechanical ventilation if needed. Hydrostatic pulmonary edema or heart failure usually occurs 1 hour after operation, often in the setting of postoperative hypertension.[32] Treatment includes diuretics, fluid restriction, dialysis if associated with renal failure, antihypertensive agents, and nitroglycerin compounds including intravenous nitroglycerin, particularly if there is ischemia associated with heart failure. If hypoxemia is uncontrollable, positive pressure ventilation with PEEP should be used and monitoring with a pulmonary artery catheter should be considered.

Significant contributions to hypoxemia occur with a low mixed venous P_{O_2} in the face of a moderate intrapulmonary shunt. The etiology of the low mixed venous P_{O_2} is usually due to a depressed cardiac output[96] and only rarely to increased oxygen consumption.[12] Theoretically, a low hemoglobin could also contribute to a low mixed venous P_{O_2} because oxygen transport will be minimal, particularly with a low cardiac output. The mechanism of the hypoxemia is related to delivery of a lowered mixed venous P_{O_2} blood directly to the arterial circulation through the intrapulmonary shunt. Treatment of the hypoxemia includes improving cardiac output to increase the mixed venous P_{O_2} and treating the etiology of the increased oxygen consumption if that is causing the low mixed venous P_{O_2}.

Pulmonary embolus, whose origin is from the deep veins of the legs in more than 90% of cases, can cause profound hypoxemia in the postoperative state. Ventilation/perfusion mismatch is the primary mechanism causing the low P_{O_2}. The incidence of fatal postoperative pulmonary embolus varies from less than 1% in patients undergoing elective general surgery[118, 122] to 3% in patients undergoing elective hip surgery.[80] Risk factors include obesity, varicose veins, malignancy, bed rest, congestive heart failure, aging, and type of surgical procedure, with prostatic and hip surgery having a higher incidence. Long-term reduction in death from pulmonary embolus should emphasize prophylaxis and prevention because many cases of pulmonary embolus are diagnosed postmortem[39] and two thirds of patients who die from the disease do so within 30 minutes after the acute event, before therapy can be started.[41] Because pulmonary embolism is merely a complication of deep venous thrombosis, prophylactic measure should be used to prevent deep venous thrombosis.[93]

Prophylaxis with low-dose heparin is usually given as a subcutaneous dose of 5000 U 2 hours preoperatively and every 12 hours postoperatively, and there appears to be no increased risk of bleeding in general surgical patients.[63] Owing to its theoretical potential for bleeding, it should not be used in patients undergoing cerebral, eye, or spinal surgery.[62] Adjusted-dose heparin therapy given as a dose of 3500 U initially and adjusted to maintain the APTT between 32 and 36 seconds is more effective than low-dose heparin in patients undergoing prophylaxis for hip or prostatic surgery.[80] If postoperative pulmonary embolism is suspected, the definitive study is pulmonary angiogram and the therapy is full-dose intravenous heparin therapy in hemodynamically stable patients.

A pneumothorax causes hypoxemia due to atelectasis and intrapulmonary shunting. The causes include percutaneous vascular cannulation, trauma, rib fractures, and mechanical ventilation.[37] Treatment of a 20% or greater pneumothorax in a spontaneously breathing patient or any pneumothorax in a mechanically ventilated patient is done with chest tube insertion and drainage. A pneumothorax of less than 20% in a spontaneously breathing patient can be watched with serial chest roentgenograms to determine the course. Clinical suspicion of a tension pneumothorax requires immediate treatment with a 14-gauge needle inserted in the second intercostal space to relieve the tension prior to chest tube insertion.

A low inspired oxygen concentration less than room air can rarely cause hypoxemia due to switching one gas for another.[48] Treatment is the administration of exogenous oxygen and discontinuation of the inappropriate gas.

Hypoxemia secondary to alveolar hypoventilation in the postoperative setting can be treated with supplemental oxygen. The etiology of hypoventilation will be discussed in the section on hypercarbia.

Diffusion impairment and hypoxemia is rare and theoretically may occur when a patient has interstitial lung disease, small lung volumes, and high cardiac outputs. In this setting, there may be insufficient time for hemoglobin to combine with oxygen with resulting hypoxemia. The treatment for this hypothetical entity is supplemental oxygen.

Determination of hypoxemia should always be made by obtaining an arterial blood gas. An initial blood gas should be compared with simultaneous noninvasive oximetric oxygen saturation readings (such as pulse oximetry) to determine that a good correlation exists. An arterial Po_2 of 65 mm Hg or greater should guarantee a hemoglobin saturation of 90% or greater. Since noninvasive oximetry is the most accurate above 90% hemoglobin saturation, the range most desired from a clinical standpoint, subsequent trends and monitoring can then be followed with oximetry.[125] Treatment of hypoxemia is usually effective by face mask unless a low arterial Po_2 is caused by a shunt of 25% or greater.[99] When hypoxemia persists despite an Fio_2 of 0.6 to 0.8 or greater, mechanical ventilation with PEEP is indicated. Ventilation with PEEP increases the FRC, decreases shunt, and eventually allows a reduction in Fio_2 to less than 50% in most patients. An Fio_2 of less than 50% can be tolerated without pulmonary complications for an extended period of time.[72]

Hypercarbia

Hypercarbia, an elevated arterial Pco_2, is due to increased production of carbon dioxide, ineffective removal of carbon dioxide (alveolar hypoventilation), or both. An elevated arterial Pco_2 per se does not define inadequate ventilation, however. Instead, a drop in pH due to a respiratory acidosis is also required because some patients are chronic CO_2 retainers.

Ineffective removal of carbon dioxide may be related to inadequate respiratory drive secondary to residual anesthetics,[73] state of consciousness,[15] metabolic alkalosis alone or superimposed on the use of narcotics,[61] and residual anesthetics and hypothermia.[103] Depressed respiratory drive may last up to 3 to 4 hours if certain opioid anesthetics are used.[13] Unless further sedatives are given in the recovery room, drug effects of this type should be gone within 4 hours. Treatment aims should be to avoid further sedatives if possible, warm up the patient, treat the etiology of the metabolic alkalosis, and allow 4 hours or more for residual anesthetic and sedative effects to wear off. Mechanical ventilation can always be continued or re-instituted to support the patient until recovery if need be.

Neuromuscular weakness causing hypoventilation can be due to inadequate reversal of muscle relaxation from insufficient administration of anticholinesterase drugs. This effect can be further aggravated with the use of antibiotics,[20, 98] furosemide, and propranolol. Patients undergoing upper abdominal surgery have postoperative diaphragm muscle weakness that may also contribute to ventilatory insufficiency and stimulate increased intercostal muscle breathing to maintain ventilation.[52] The addition of morphine in this state will diminish the action of the intercostal muscles, making ventilatory insufficiency even more likely.[106] The passage of time, adequate reversal of muscle relaxation, avoidance of interacting drugs, minimal sedation, correction of electrolyte imbalance due to hypokalemia, hypomagnesemia, and hypocalcemia should help treat the neuromuscular weakness. Intravenous aminophylline may help postoperative upper abdominal surgery patients by improving diaphragmatic dysfunction with an aminophylline level between 10 to 15 $\mu g/mL$.[44]

Increased dead space ventilation from respiratory therapy equipment, pulmonary embolic disease,[46] pulmonary hypotension,[69] and intrinsic lung disease can increase the work of breathing to maintain a given P_{CO_2} and eventually cause alveolar hypoventilation and hypercarbia. Treatment of the underlying disease and respiratory equipment modification should hopefully decrease the dead space enough to decrease the work of breathing.

Reduced pulmonary compliance due to a surgical insult (thoracic or abdominal surgery), obesity,[14] preoperative or acquired (ARDS) restrictive lung disease, and postoperative heart failure[117] can markedly increase the work of breathing, cause respiratory muscle fatigue, and lead to CO_2 retention. Sitting up in bed and IS can help improve pulmonary compliance in all postsurgical patients, particularly the obese. If treatment of heart failure and lung disease is unsuccessful and respiratory failure supervenes, positive pressure mechanical ventilation with CPAP will markedly improve pulmonary compliance and decrease the work of breathing until the patient stabilizes.[68, 116]

Increased airway resistance can cause hypercarbia by increased CO_2 production through an increased work of breathing and by decreasing CO_2 clearance. The etiology is usually secondary to chronic obstructive lung disease, asthma, or heart failure, although upper airway obstruction is possible. Treatment with β-agonists, cholinergic agents,[4, 104, 134] and aminophylline should improve the noncardiac bronchospasm. Heart failure is treated with fluid restriction, oxygen, diuretics, and so forth.

Increased CO_2 production can occur in the postoperative state due to shivering, recovery from operative hypothermia,[123] malignant hyperthermia,[58] or rapid intravenous hyperalimentation.[5, 7] When considering the multitude of other causes of decreased clearance of CO_2 in the recovery room, even a mild augmentation in CO_2 production might precipitate ventilatory insufficiency. Treatment includes changing[6] or discontinuing hyperalimentation, dantrolene, rapid cooling, fluid therapy, and other symptomatic support for malignant hyperthermia.[57]

Unlike oxygen saturation monitoring, no reliable noninasive arterial P_{CO_2} monitor is available at this time.[138] Therefore, arterial blood gas analysis is needed to determine the partial pressure of carbon dioxide. End tidal P_{CO_2} can be monitored when technically feasible.

Airway Obstruction

Signs of airway obstruction include flaring of the nostrils, retracting of the rib cage and suprasternal notch, stridor, and decreased breath sounds. Slightly more objective measures of determining air movement include listening and feeling the exhaled air. Use of a ventimeter can also be used to measure the volume of air moved.

The most common postoperative cause of upper airway obstruction is pharyngeal obstruction from a sagging tongue in the unconscious patient.[111, 112] Three maneuvers, backward tilt of the head, anterior displacement of the mandible, and closure of the mouth, have been determined to be effective in relieving the obstruction.[90] The most effective maneuver is backward tilt of the head. If these maneuvers are ineffective, lateral positioning and a nasal or oral airway can be inserted. The nasal airway is preferred because the oral airway may stimulate vomiting, gagging, and occasionally laryngospasm. If all these measures are unsuccessful, endotracheal intubation can be performed.

Laryngospasm,[133] tight apposition of the vocal cords, is another possible etiology of airway obstruction. It usually occurs during emergence from anesthesia, when the larynx is irritated by secretions, foreign matter, or pain. Depending on the cause, treatment should include suctioning or removing foreign material from the oropharynx, 100% oxygen, removing any painful stimulus, applying positive pressure to the airway, and forward jaw thrust. If these steps are not fruitful, a short-acting muscle relaxant (succinylcholine, 0.1 mg/kg) allows relaxation for assisted lung ventilation. When all else fails, endotracheal intubation under direct laryngoscopy can be performed. In the rare instance that endotracheal intubation is unsuccessful, emergency tracheostomy or cricothyroidotomy should be performed. Cricothyroidotomy using a 14-gauge intravenous catheter can be performed with the administration of low-flow 100% oxygen at 3 L/minute[124] until the airway can be secured.

Aspiration Pneumonia

Aspiration into the lungs from the mouth or the stomach is a common occurrence in everyday life. Several investigators[2, 22] have put dye or contrast material into the mouths of tracheotomized or sleeping patients and noted pulmonary aspiration with no clinical sequelae. Other investigators have instilled dye into the stomach of patients preoperatively and found the dye in tracheal aspirates with minimal pulmonary complications.[16, 38]

Aspiration of matter in the perioperative period can cause significant postoperative pulmonary dysfunction and infection depending on the quantity, frequency, and type of aspirate. Aspiration of solid particulate matter, usually occurring in children 1 to 3 years of age,[84] included peanuts, vegetable matter, inert metal, meat bolus, plastic items, popcorn, and other miscellaneous items. Initial symptoms include cough and bronchospasm with subsequent localized atelectasis and pneumonia. Treatment includes foreign body removal by coughing or bronchoscopy, pulmonary toilet, and antibiotic treatment with penicillin if pneumonia occurs. Removal of foreign material must include surgical removal if bronchoscopy is unsuccessful in order to clear up the pulmonary infection. Retention of aspirated vegetable matter is particularly troublesome and causes a chronic granulomatous reaction similar to miliary tuberculosis.[143]

Aspiration of nontoxic fluids such as water, saline solution, barium, and neutralized gastric contents usually causes only transient respiratory distress. Treatment includes immediate tracheal suctioning and support. If no persistent symptoms or pulmonary infiltrate develops, no other treatment is necessary.[9]

Acid aspiration syndrome was first described by Mendelson[86] in 1946 in obstetric patients. He suggested that antacids might be beneficial if used prophylactically in these patients. Other investigators noted it took a gastric pH less than 2.5 to develop significant tissue damage with aspiration of fluids into the lungs.[56, 135] In 1974, Roberts and Shirley[109] noted that the maximum volume of gastric aspirate at a pH of less than 2.5 that did not produce significant pulmonary changes in rhesus monkeys was 0.4 mL/kg. The equivalent gastric volume in humans is about 25 mL. From this came the minimum amount of fluid that could be in the stomach at a pH less than 2.5 that should not cause pulmonary damage if aspirated. Roberts and Shirley then noted that giving particulate antacids (magnesium and aluminum hydroxide) within 4 hours of inducing anesthesia in obstetric patients increased the gastric pH without changing the volume, therefore reducing the risk of acid aspiration. It was soon noted both in dogs[53] and humans[17, 147] that aspiration of neutral antacids caused serious pulmonary dysfunction and death with chronic granulomatous changes noted in the dog lung preparations. With this knowledge a shift was made to nonparticulate antacids such as sodium citrate, preoperative histamine-2 (H2) antagonists, and blockers of hydrogen ion secretion such as omeprazole.

A further study done in rats in 1984 revealed that a gastric content of less than 0.4 mL/kg with a gastric pH less than 1.0 resulted in death 90% of the time when instilled into the lungs.[66] Once the pH was greater than 1.8, however, the mortality dropped precipitously, even with gastric volumes greater than 2 mL/kg. This brought about the concept that pH was much more important than the critical volume, which may not exist. No such study has obviously even been done in humans to corroborate the rat extrapolation.

Studies with cimetidine in a variety of preoperative and emergency circumstances reveal an improvement in gastric pH with some treatment failures in every group.[29, 30, 40, 128] One study used cimetidine, 200 mg intramuscularly, 50 minutes prior to emergency cesarean section in 100 patients. Just prior to induction of anesthesia, 30 mL of 0.3 M sodium citrate was administered orally. Analysis of gastric aspirates found an average gastric pH greater than 6.0 with no pH less than 2.5.[136] Ranitidine (150 mg) versus cimetidine (300 mg) in a double-blind comparison given orally 2 or 4 hours prior to surgery showed fewer treatment failures with ranitidine and a significant reduction in volume of gastric aspirate with ranitidine (less than 10 mL).[91] Famotidine[23] is a new H2-receptor antagonist with a potency 20 times that of cimetidine and 7.5 times that of ranitidine. Comparison of famotidine with ranitidine in a randomized double-blind study in patients undergoing elective surgery found no treatment failures in either group.[47] The pH was 7.0 or greater in both groups (average 2.8 in controls) with a statistically significant decrease in gastric volume in both groups compared with the control group. In addition, famotidine significantly decreased the gastric volume compared with ranitidine. If famotidine proves to be valid and is administered prior to emergency surgical procedures, Mendelson's syndrome may become a syndrome of the past.

Omeprazole,[27] a substituted benzimidazole that blocks gastric acid secretion by inhibiting the parietal cell proton pump, had a 15% failure rate in maintaining a gastric pH less than 2.5 when used preoperatively for elective cesarean sections.[89]

The general recommendations for prevention of aspiration include delaying surgery for 8 to 12 hours in anyone who has eaten solid food, use of H2-histamine receptor blockers 2 to 4 hours preoperatively in high-risk patients (pregnant, obese, emergency surgery patients, history of hiatal hernia or reflux esophagitis), administration of nonparticulate antacids in high-risk aspiration groups (especially if there is no time to give H2 blockers), and awake oral or nasal endotracheal intubation or "rapid sequence" approach in high-risk patients. Metoclopramide, an antiemetic agent that accelerates gastric emptying and increases lower esophageal sphincter tone, may be of benefit in the parturient with a full stomach. Studies regarding its use show mixed results.[28, 151]

Patients at risk for aspiration in the postoperative period, including the obtunded, should not be extubated until all residual effects of anesthetics and narcotics have worn off. Suspicion of aspiration secondary to a vomiting patient, new fever with a pumonary infiltrate, and hypoxemia with new pulmonary infiltrate should be monitored and sputum obtained for gram stain and culture. A witnessed aspiration should not be treated empirically with antibiotics unless clinical infection is apparent because bacterial infection does not necessarily occur with all aspirations. If clinical infection is present, therapy should be directed by sputum gram stain and sputum and blood cultures. When the sputum is inconclusive and clinical infection is apparent, empiric antibiotic therapy should cover hospital-acquired aspiration pneumonias in which anaerobes, *Staphylococcus aureus*, and gram-negative rods are prominent.[8, 67] Steroids are not indicated for the treatment of aspiration pneumonia and may even be harmful in some septic patients.[19]

SUMMARY

Postoperative ventilatory and pulmonary complications have been present since the first operation but the ability to monitor, detect, and prevent some of these conditions is improving rapidly with the advent of routine noninvasive oximetry in the PACU. As the realization sets in that hypoxemia is a common event in the PACU, more aggressive preventive measures can be taken to prevent its occurrence. The treatment of Mendelson's syndrome has evolved rapidly in the last few years with the use of more powerful and sophisticated H2 blockers such as ranitidine and famotidine. Both drugs increase gastric pH and decrease gastric volume. It is hoped that in the not too distant future Mendelson's syndrome will be a syndrome of the past.

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The Obstetric Recovery Room

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As the overall involvement of anesthesiologists in the delivery of both anesthesia care and intensive care for obstetric patients grows, the importance of assuring the adequacy and ability of the obstetric recovery room to handle postanesthesia obstetric patients likewise increases. There are several relatively unique characteristics of both the obstetric patient and obstetric surgical procedures that impact on recovery room considerations. First, patients taken care of in labor and delivery suites are generally considered to be at relatively low risk for postoperative complications, since they are usually young and healthy patients. Second, there is an increased use of major regional anesthesia in obstetric patients relative to patients taken care of in other operating rooms and recovery rooms in a hospital. Third, several diseases and operations are unique to obstetric practice. Fourth, several special postoperative pain relief techniques are being used increasingly among postcesarean section patients. Fifth, the labor and delivery suite and the obstetric recovery room are frequently geographically separate from the general operating rooms and recovery room in many hospitals. Finally, a concept that appears to be growing in popularity is the combination of previously physically separate labor, delivery, recovery, and postpartum (LDRP) rooms into one private room for individual patient care. The impact of each of these special considerations unique to the obstetric recovery room will be considered individually, so that rational training, planning, and policies can be instituted to deliver the safest possible medical care.

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THE PERCEPTION OF OBSTETRIC PATIENTS BEING LOW RISK

Since most patients undergoing labor and delivery are young and usually healthy, they will usually be classified as American Society of Anesthesiology (ASA) class I patients, excluding considerations of the pregnant state itself. For this reason, it is commonly extrapolated that obstetric patients in general will require a less intensive level of care than the average patient in other operating and recovery room environments within the same hospital. As we shall discuss, this perception may or may not be true depending on the individual obstetric case mix in a given hospital. A common result of this perception is, however, that hospital administrators frequently give low priority to staffing obstetric recovery rooms with personnel in quantity and quality similar to other recovery areas in the hospital. For example, labor and delivery nurses are almost always rotated through the recovery area, with a majority of their time spent caring for laboring patients. This lack of a "dedicated" recovery room staff, well versed and familiar in recovery room nursing techniques, is very different from staffing patterns of other recovery areas. The number of nurses assigned to recovery room duties in the labor and delivery suite is also usually less than that assigned to other recovery rooms, especially when peak labor and delivery patient volumes are considered. However, the Joint Commission on Accreditation of Health Care Organization (JCAHO) regulations clearly state that the standards of nursing care should not vary for similar patients between different postanesthesia recovery areas within a given hospital.[1] An implication of this policy is that if patients who are greater than ASA category II are to be taken care of in an obstetric recovery room, and/or the complexity of obstetric surgery is equal to that of surgery performed, for example, in the gynecologic operating rooms, then the recovery room nursing standards should be similar for those two areas. Under current patterns of staffing and monitoring in many hospitals, however, ASA I or II patients following an intra-abdominal operation (i.e., a cesarean section) in the labor and delivery suite will be given "less intensive" postoperative care than patients following a hysterectomy in a gynecologic operating area. This occurs in contradiction to the fact that patients in the immediate postpartum period (for reasons outlined in the following discussion) will frequently be undergoing physiologic and hemodynamic changes that make them *more* susceptible to physiologic derangement in the recovery room.

Other forums have also addressed the issues of uniformity of staffing for all recovery rooms. The American Society of Post-Anesthesia Nurses (ASPAN) has published a series of standards for postanesthesia nursing care.[3] Included in these standards are recommendations for

Table 1. *Recommended Patient-to-Nurse Ratios*

RATIO	REMARKS
3:1	Awake and stable, uncomplicated patients
2:1	Stable, unconscious patients Patients following major surgery whose systems have stabilized
1:1	Patients requiring life support care

From Standards of Nursing Practice. American Society of Post-Anesthesia Nurses, Richmond, VA, 1984.

appropriate maximum patient-to-nurse ratios in a postanesthetic recovery area. The maximum patient-to-nurse ratio recommended is 3 to 1, and that only applies to stable awake uncomplicated patients (Table 1). The implication of this recommended ratio is that for any but the smallest labor and delivery areas there should be at least one and possibly two or three nurses assigned concurrently to recovery room responsibilities. The standards go on further to state that "it is recommended that two licensed staff nurses, one of whom is an RN, be present whenever a single patient is recovering from anesthesia." Staffing according to this latter recommendation would appear to encourage a minimum of two staff nurses in every recovery room whenever patients are present. This minimum staffing would eliminate a commonly occurring problem arising in some obstetric recovery rooms when the assigned nurse is also responsible for the transfer of patients to the postpartum floor. When this occurs, the recovery room is left unstaffed during patient transfer. It should be emphasized that the recommendation of a 3 to 1 ratio is merely the *maximum* ratio acceptable for low-risk patients; frequently, more intensive nursing care may be necessary if higher risk patients are present.

The ASA Standards for Postanesthesia Care approved in 1988 extend the JCAHO requirement for standardized nursing care in all recovery areas to include standardization of *all* aspects of postanesthesia care.[4] The ASA Standards are prefaced with the statement that "these Standards apply to postanesthesia care in all locations," and go on to endorse the ASPAN and JCAHO Standards.

This becomes important when monitoring capability in the obstetric recovery room is concerned. Because of the low-risk perception of obstetric patients, there is also a tendency to divert the allocation of resources for the acquisition of monitoring equipment away from the obstetric recovery room toward other recovery areas. Although the ASA Standards do not closely delineate monitoring methods, most departmental guidelines for monitoring capability in the recovery room now include automated blood pressure devices that automatically measure blood pressure, electrocardiographic (EKG) monitors, and pulse oximeters. The use of such equipment, if not already becoming available in nearly all obstetric recovery rooms, will soon become widespread. For reasons outlined, the immediate availability of both these types of monitors to recovering patients in obstetrics is occasionally essential. Within each institution, uniformity of monitoring capability in each recovery room should be the goal.

THE WIDESPREAD USE OF MAJOR REGIONAL ANESTHESIA

In many hospitals, over half the patients who undergo labor and delivery will have a major regional anesthetic (spinal or epidural) administered during the course of labor and delivery. The implication of this increased use of regional anesthesia is that the obstetric recovery room staff should be among the best trained and most experienced in the hospital in the care of patients recovering from major anesthesia. To achieve such expertise, ongoing in-service training by the anesthesiology staff should be made readily available to recovery room nurses. Such training is especially important during nursing orientation to the obstetric recovery area and should include the underlying physiology of hemodynamic changes during spinal and epidural anesthesia, detection of sensory and motor level of blockade, the implications of motor and sensory blockade, and protocol for detection and treatment when problem arise. The importance of achieving nursing expertise in managing postregional anesthesia patients is illustrated by the following example. Although it seems more humane to administer an intramuscular medication within an area of persistent residual sensory blockade in a patient with a resolving spinal or epidural anesthetic, this practice may be hazardous, since parathesias may not be elicited if the needle is in or near a nerve when injection occurs. The result of such a practice will be occasional patients who complain of a small area of residual numbness postoperatively, corresponding to the location in which the intramuscular injection was given. Only through in-service training and quality assurance and risk management feedback can the nursing staff be made aware of such complications and develop policies and procedures to avoid recurrence.

The obstetric recovery room is also the recovery room in which patients will be routinely watched following a relatively high (T_1–T_4) spinal or epidural anesthetic, since such levels are frequently sought when administering major regional anesthesia for cesarean section, and occasionally occur unintentionally even during analgesia for labor and delivery. In fact, obstetric anesthesia is one of the few areas in anesthetic practice in which this high an anesthetic level is routinely sought for surgery. When these patients arrive in the recovery room they must be closely observed for the sequelae of relatively high spinal and epidural anesthesia. Again, only through in-service training and experience will the nursing staff become familiar with the safe care of patients with relatively high spinal or epidural block.

DISEASES AND SURGICAL PROCEDURES UNIQUE TO OBSTETRICS

Dilatation and Curettage Following Miscarriage

The special needs in the postoperative period of the patient following dilatation and curettage for a missed or spontaneous abortion need to be considered. In most hospitals, these patients are sequestered after their procedure and not allowed to recover in the recovery room with other mothers and their newborn babies. This is usually done in the hope that isolation of patients following dilatation and curettage will avoid development of a depression reaction following exposure to the normal obstetric recovery room environment. The medical problem with this practice of separation from other recovering patients is that the patients frequently will not only be segregated from the other recovery patients but will be placed in a labor room by themselves to recover. If the procedure was done with either relatively heavy sedation, general anesthesia, or both, these patients are at increased risk for respiratory problems during emergence from anesthesia and need to be closely monitored. More intensive nursing care than usual may be necessary; perhaps even a 1 to 1 patient nurse ratio may be indicated during the recovery period, at least until the patient is awake and alert enough so that most risk of significant respiratory depression is past. The ASPAN standards mentioned previously[3] could even be interpreted to mandate such staffing if the patient is truly isolated from other nursing care areas. Transcutaneous monitoring of P_{CO_2} and/or P_{O_2}/SaO_2 may also be desirable in these patients to detect respiratory compromise.

Cervical Suture

The placement of a cervical suture for cervical incompetency is a procedure unique to obstetric surgery. During recovery, patients are frequently maintained in Trendelenburg's position for at least several hours. They are also monitored for spontaneous uterine activity, which may normally follow any procedure in which the uterus or cervix are manipulated. When significant uterine contractions occur, intravenous tocolytic therapy may become necessary. Ritodrine is currently the only FDA-approved drug for intravenous tocolysis. The complications of intravenous administration of ritodrine include hypokalemia, hyperglycemia, tachycardia, myocardial ischemia, congestive heart failure, and a dilutional anemia. Patients receiving intravenous ritodrine following cervical suture placement should be monitored for the presence of all of these side effects.

Postpartum Hemorrhage

One obstetric complication that arises in the recovery room that deserves special attention is postpartum hemorrhage. Of all obstetric complications, this is the one in which morbid or mortal outcome is perhaps most easily avoided, given prompt detection and definitive treatment. Although postpartum hemorrhage is most frequently detected while still in the delivery room, it can certainly occur in the several hours (and even days) following delivery. For this reason, the postpartum patient should have her vital signs monitored frequently if more than normal bleeding has occurred during vaginal delivery or cesarean section. In addition, the amount and characteristics of vaginal bleeding should be noted at regular intervals during the recovery room stay. Even in the absence of large amounts of vaginal bleeding the diagnosis of postpartum hemorrhage should immediately be considered and investigated if any unexplained hypotension occurs. The importance of *immediate* detection and treatment of postpartum hemorrhage cannot be overemphasized, since hypotension itself causes hypotonia of the uterus, which may increase the bleeding, resulting in compounded hypovolemia and severe hypotension. Therefore, whatever monitoring is used during recovery must be able to detect the early signs of hypovolemia in the postpartum patient. Blood pressure should be measured and observed at frequent intervals. An automated blood pressure device with appropriate alarm settings may be useful. Heart rate should be monitored for unexplained tachycardia. The patient's state of consciousness should be observed for changes consistent with cerebral hypoperfusion. Finally, the amount of external blood loss needs to be observed and measured.

The medical treatment of postpartum hemorrhage is twofold: (1) maintenance of blood pressure at levels compatible with survival of the patient, and (2) delineation and treatment of the underlying cause. The most common techniques used to maintain blood pressure are the rapid infusion of intravenous fluids, the administration of vasopressors, and the use of Trendelenburg's position. All patients following even uncomplicated delivery should have intravenous access available because of the risk of postpartum hemorrhage, for the administration of intravenous fluids, vasopressors, and ecbolic agents. The preference to avoid antepartum administration of alpha-adrenergic agonist vasoconstrictors as therapy for hypotension is no longer a consideration postpartum, and agents such as phenylephrine play an increasingly important role. Because hypovolemic patients will frequently be tachycardic as well, the use of ephedrine or epinephrine for the treatment of hemorrhagic hypotension may not be as desirable as phenylephrine to attain resolution of hypotension while maintaining a reasonable heart rate. Occasionally, epinephrine will be necessary to maintain blood pressure if the hemorrhage is severe and the patient is unresponsive to ephedrine or phenylephrine. The airway should be protected if consciousness is compromised as the result of hypotension.

Concomitant with attempts at restoring blood pressure, efforts should be undertaken to diagnosis and treat the underlying cause of the bleeding. In most cases, uterine atony is the cause of bleeding. Uterine atony can be treated by uterine massage and the administration of ecbolic agents, such as methylergonovine, oxytocin, or prostaglandin $F_{2\alpha}$. The usual dose of methylergonovine is 0.2 mg administered intramuscularly. Methylergonovine can also be given intravenously, with caution and observation for hypertension. When administered intravenously, it should be given in a dose of 0.02 mg every minute until either uterine tone has improved or blood pressure begins to rise as a result of a direct vasoconstrictive effect. Ueland[5] recommends the use of prostaglandin $F_{2\alpha}$ instead of ergot alkaloids because of this risk of hypertension. Oxytocin should be used with caution in patients who are hypotensive and hypovolemic, since intravenous administration of oxytocin results in a dose-dependent decrease in systemic vascular resistance. Doses as small as 200 mU may drop blood pressure significantly,[2] a side effect that is certainly unwelcome, if not dangerous, in the presence of pre-existing hypotension.

If surgical intervention is necessary to treat the cause of bleeding, it should be undertaken as soon as possible. Frequently, a vaginal examination will be conducted first to ascertain whether or not the the bleeding originates from the cervix or the vagina. If it does not, then a laparotomy may need to be performed, and the patient must be appropriately prepared for surgery. For this reason, patients in the recovery room, even after normal spontaneous vaginal delivery, should not be given anything by mouth until it is unlikely that postpartum hemorrhage would be a problem.

Toxemia of Pregnancy

Approximately 5% of pregnant patients will have the diagnosis of pregnancy-induced hypertension or toxemia of pregnancy. These patients remain at increased risk for developing complications of toxemia even in the postpartum period. For example, pre-eclamptic patients can experience eclamptic convulsions for up to 48 hours following delivery and, in rare cases, even later. Patients who exhibit any signs of toxemia of pregnancy such as hypertension, proteinuria, edema, especially with accompanying hyper-reflexia, abdominal pain, headache, visual scotomata, confusion, or thrombocytopenia should be observed closely in the recovery room for worsening. Patients in whom the diagnosis of pre-eclampsia is confirmed should be given appropriate anticonvulsant therapy, which may include intravenous magnesium sulfate, phenytoin, or diazepam. Such patients should be monitored closely postpartum while receiving anticonvulsant medications. In our institution, these patients are monitored for 24 hours in the labor and delivery suite before being discharged to the regular postpartum floor.

When a convulsion occurs in the recovery room it should be immediately treated and an etiology investigated. If it is an eclamptic convulsion, then it is typically short-lived and self-resolving. Patients may exhibit a postictal state but rarely need their airway or ventilation supported following the seizure. If any vomiting or regurgitation has occurred during the convulsion, appropriate evaluation for aspiration pneumonitis should be performed and oxygenation should be monitored.

Since subarachnoid hemorrhage is a leading cause of death in eclamptic patients, severe hypertension (diastolic blood pressure > 110 mm Hg) should be aggressively treated. Hydralazine, labetalol, or calcium channel blockers will frequently be adequate. A potent rapid-, short-acting agent such as nitroprusside may become necessary. Direct invasive arterial monitoring should be instituted if nitroprusside is used, and may even be desirable in any severely toxemic patient.

Pulmonary edema can occur following delivery of toxemic patients. This is probably secondary to the increased extravascular fluid volume being returned to the central circulation following delivery. Occasionally, myocardial dysfunction is also present. Intravenous fluids should be used sparingly following delivery unless there is good evidence of continuing intravascular volume depletion and no impairment of oxygenation. Frequent examinations of the lungs may be useful, as well as SaO_2 monitoring. Patients with severe pre-eclampsia should also be monitored for the development of the "HELLP" syndrome, which consists of *h*emolysis, *e*levated *l*iver enzymes, and thrombocytopenia (*l*ow *p*latelets), because this syndrome is associated with a much higher morbid and mortal outcome.

UTERINE OBSTETRIC DRUGS

There are two general catagories of pharmacologic agents that are used almost exclusively in obstetrics: ecbolics and tocolytics. The ecbolic agents most frequently used in the postpartum period include

Table 2. *Ecbolic Drugs*

DRUG	DOSE, ROUTE	COMMENT
Oxytocin (Pitocin)	≤100 mU/min IV infusion	Bolus or rapid infusion may cause hypotension
Methylergonovine (Methergine)	0.2 mg IV 0.02 mg IV every min	May cause hypertension and arterial spasm. Give slowly IV, in small increment if necessary
Prostaglandin $F_{2\alpha}$ Carboprost (Hemabate)	250 μ IM or into myometrium. May be repeated every 15–90 min	May cause bronchospasm and hypertension. Can be repeated if necessary

Abbreviations: IV = intravenously; IM = intramuscularly.

synthetic oxytocin, methylergonovine (an ergot alkaloid), and prostaglandin $F_{2\alpha}$ (Table 2). Oxytocin is usually administered routinely following vaginal delivery to promote uterine contractility and decrease the risk of postpartum hemorrhage. Oxytocin is frequently administered as an infusion. Since intravenous boluses as small as 200 mU may cause hypotension,[2] oxytocin should never be given as a bolus injection (especially in the setting of postpartum hemorrhage), but should always be administered as a relatively slow infusion. Methylergonovine is another agent that promotes uterine contractility. Methylergonovine is usually given intramuscularly but can be given in small incremental intravenous doses with caution. Prostaglandin $F_{2\alpha}$ can also be used to contract the uterus postpartum. The usual dose is 250 μg either given intramuscularly or as direct injection into the myometrium in multiple sites.[5] It may be repeated.

Tocolytics that are commonly used include ritodrine, terbutaline, and magnesium sulfate. The side effects of terbutaline are similar to those of intravenous ritodrine, which have been previously mentioned. Magnesium sulfate is occsionally used as a tocolytic agent and can have profound cardiac and respiratory side effects if given in large doses. It is therefore recommended that patients receiving this drug have serum Mg^{2+} levels checked regularly.

MEDICAL PROBLEMS OF NEWBORNS

Newborns can have medical problems in the immediate neonatal period, and because newborn infants will frequently accompany the mothers into the recovery room, personnel taking care of the mothers must also serve as provider to the infant. The foremost medical problems in the newborn period requiring attention are (1) hypothermia due to poor thermal regulation in the infant, especially in cold environments; (2) hypoglycemia due to stress combined with the relatively small glycogen stores of the neonate; and (3) impairment of the normal physiologic changes occurring in the newborn's cardiorespiratory system during transition from fetal to neonatal life. Most frequently, if the infant is dried in the delivery room and kept wrapped (and not exposed to ambient air), thermal regulation is not a problem. Likewise, if no undue stress has occurred during delivery and the mother is neither diabetic nor has received a large amount of intravenous glucose toward the end of her labor, hypoglycemia is rarely a problem in the newborn. However, cardiorespiratory problems, especially problems with oxygenation, can occur in any term infant in the immediate neonatal period. The most frequent etiologies would be (1) meconium aspiration leading to meconium pneumonitis, (2) amniotic fluid aspiration leading to amniotic fluid pneumonitis, and (3) transient tachypnea of the newborn (TTN), a diagnosis given to infants with mild respiratory distress when no other cause can be found. The symptoms of all of these three processes will be similar and include signs of mild to moderate respiratory distress such as flaring of the nose, grunting, paradoxical movement of the chest wall during breathing, and generalized cyanosis. If infants exhibit any of these signs, then the recovery room staff should arrange a transfer to the neonatal nursery for more careful observation and physiologic measurements. Because many obstetric nurses may not be well versed in the diagnosis of newborns, in-service training in the care and problems of newborns should be conducted for any nurse who is taking care of obstetric recovery room patients.

SPECIAL POSTOPERATIVE PAIN TECHNIQUES

Increasingly popular methods for post-cesarean section pain relief are the epidural administration of narcotics and the use of patient-controlled analgesia (PCA), either intravenously or epidurally. Since these are relatively new techniques of postoperative pain relief, the anesthesiology staff initiating these techniques in the recovery room should be certain that the recovery room nursing staff is adequately trained in the use of these techniques in general, and the use of infusion or PCA pumps specifically. Adjustments of the pain control regimens frequently become necessary in the recovery room, and protocols should be developed for the nursing staff to make such adjustments if the anesthesiology staff is unavailable to do so. As with the administration of any narcotic, appropriate monitoring protocols should be instituted when patients receive either intraspinal or intravenous narcotics.

PHYSICALLY SEPARATE OBSTETRIC AREA

In many hospitals the labor and delivery area is physically separated from the main operating rooms. Therefore, anesthesiologists may only be called to the labor and delivery area when a procedure becomes necessary and may need to return to the general operating rooms after the procedure has been performed. This physical separation will occasionally result in the unavailability on an immediate basis of the anesthesiology staff to handle obstetric recovery room problems. When practicing in such a setting, anesthesiologists should transfer the care of postpartum patients to the recovery staff only when the need for further intervention by an anesthesiologist appears remote. For example, patients with questionable reversal of neuromuscular blockade following general anesthesia for cesarean section should remain accompanied by an anesthesiologist or nurse anesthetist until all question of the adequacy of reversal is gone. The obstetric recovery nursing staff should be aware that it may also take slightly longer than usual to obtain the assistance of the anesthesiology staff when required. If the anesthesiologist must return to the main operating room areas and the patient is potentially unstable, then transfer of the patient to the main recovery room (where anesthesiology assistance is more immediately available) should be considered.

THE COMBINED LABOR, DELIVERY, RECOVERY, AND POSTPARTUM ROOM

An increasingly popular trend in the design of obstetric areas is the concept of physically combining labor, delivery, recovery, and postpartum care areas into one private room. In uncomplicated cases, the laboring patient would therefore spend her entire hospital course in the same room. Since postanesthesia care may need to be delivered in such rooms, the design and staffing of these rooms should be planned with the anesthesiology care team. Nurse staffing and monitoring capability should be within standards proposed for any other recovery areas in the hospital. If this can be accomplished, safe postoperative care can be delivered in such a setting. Unfortunately, an equivalent level of staffing monitoring will probably require additional allocation of nursing and equipment resources, since in the traditional recovery room settings such resources can be shared among patients to some extent. Although the temptation exists to minimize the amount of gadgetry present in such rooms and to minimize healthcare provider-to-patient contact, the immediate postpartum period is, for reasons mentioned previously, a critical period in terms of potential morbid complications such as postpartum hemorrhage. Equipping and staffing LDRPs must take this into consideration.

SUMMARY

The patients being cared for in an obstetric recovery room are not always uncomplicated, ASA I patients. Staffing and monitoring capability of the obstetric recovery room should reflect the same standards used in other recovery rooms in the hospital. The unique nature of several obstetric diseases, procedures, and drugs should be well known and understood by those caring for postpartum patients.

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Management of Postanesthetic Pediatric Problems

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Anyone involved in the postanesthesia care of infants and children should be familiar with the anatomic and physiologic differences of these patients compared with adults and understand the emotional needs and psychological problems of these patients and their parents.

RESPIRATORY COMPLICATIONS

Although the difference in size is the most striking feature when comparing pediatric and adult patients it is the difference in the anatomy of the airway that is responsible for most of the problems encountered in recovering from general anesthesia. Although the actual size and weight are much smaller, the body-surface-to-weight ratio increases as size diminishes. The result is a much greater propensity for heat loss. Besides the actual difference in size, there is also a difference in the relative proportion of body structures. For instance, the newborn has a relatively large head, with a small thorax and a protuberant abdomen. Respiratory complications cause most of the postanesthetic problems seen within the first hour following the end of anesthesia. They include (1) airway obstruction, (2) postextubation croup, (3) respiratory depression, (4) aspiration, (5) apnea, and (6) acute respiratory failure.

Airway Obstruction

The anatomic configuration of the pediatric airway predisposes to upper airway obstruction and hypoventilation. The main characteristics are the following:

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1. The tongue is large relative to the size of the oral cavity and located closer to the roof of the mouth.
2. The larynx is positioned approximately one interspace higher (C3–C4) in the neck when compared with the adult (C4–C5). The more immature the infant, the higher the position of the larynx.
3. The epiglottis is narrow and omega-shaped; it is tilted posterior to the axis of the trachea.
4. The vocal cords are slanted upward and posteriorly. The loose alveolar tissue of the glottis is prone to edematous swelling with minor trauma or overhydration.
5. The narrowest portion of the infant larynx is the nondistensible cricoid ring, the only circumferential cartilaginous structure. This feature persists until puberty when cricoid and thyrocartilage grow. Thereafter the rima glottidis becomes the narrowest part of the larynx.[38]
6. The ribs of the infant are cartilaginous and the configuration of the rib cage tends to be circular rather than ellipsoid as in the adult.[67] The horizontal position of the ribs prevent the "bucket handle" effect of intercostal muscle contraction.
7. The angle of insertion of the diaphragm is almost horizontal instead of oblique as in the adult, which results in decreased efficiency of contraction.[87] In addition, the diaphragm contains less type I (slow twitch, high oxidative) fatigue-resistant muscle fiber in the newborn and young infant.[108]
8. The lungs of the newborn and infant contain less elastic elements, and the result is a higher specific compliance, which is compliance standardized to lung volume (functional residual capacity, vital capacity, or total lung capacity) or greater distensibility of the infant lung.[1] The extremely compliant rib cage contributes little to maintenance of negative intrathoracic pressure and lung volumes. This together with the lack of elastic recoil in the lung tissue leads to collapse of small airways and increased closing capacity.[76]

Although the abdominal contents in the upright position act as a pull on the diaphragm, increasing lung volumes, they tend to push the diaphragm cephalad in the supine position in a nonuniform pattern, resulting in a loss of functional residual capacity.[12, 14] Soft-tissue obstruction of the upper airway occurs during general anesthesia, in deep sleep stages, in patients with apnea-hypersomnia, and in sleeping infants whose necks are slightly flexed.[123] Owing to relaxation of the pharyngeal musculature, the tongue is displaced posteriorly and the negative pressure of inspiration results in airway collapse.[96] Extension of the neck decreases the tendency to airway closure; however, positive pressure is required to reopen the collapsed airway.[123] Repeated upper airway collapse may be treated with low-level continuous positive airway pressure (CPAP).[123] Insertion of an oropharyngeal airway may alleviate the problem until the patient is fully awake.

Obstructive sleep apnea may be present immediately postoperatively in as many as 10% of patients undergoing cleft palate surgery.[107] The incidence of airway obstruction is particularly high in patients with Treacher Collins syndrome, hemifacial microsomia, and Stickler syndrome.[107]

Other common causes for postanesthetic upper airway obstruction in the pediatric age group include enlarged tonsils and adenoids, laryngeal spasm, laryngeal edema, and swelling due to surgery of the airway and surrounding tissues.

The hallmarks of airway obstruction are retractions (suprasternal, subcostal, intercostal), nasal flaring, inspiratory stridor or crowing, and decreased or absent air entry.[108]

Airway obstruction should be relieved promptly. With complete cessation of gas exchange, arterial carbon dioxide tension rises between 10 and 12 mm Hg in the first minute, between 7 and 10 mm Hg in the second minute, and between 3 and 6 mm Hg during the third minute in children. Oxygenation rapidly declines. A starting Po_2 of 480 mm Hg drops to 80 mm Hg within 3 minutes.[46] Airway obstruction due to pharyngeal obstruction by the tongue can be effectively overcome by hyperextension of the head with anterior displacement of the mandible.[86] If obstruction is still not relieved, a nasal or oral airway can be inserted. The nasal airway is preferred because it is better tolerated. The oral airway may stimulate gagging, vomiting, and laryngospasm. One hundred per cent oxygen should be administered by face mask until the obstruction is relieved. Besides soft-tissue obstruction, airway obstruction because of laryngeal spasm is frequently seen in the early postanesthetic period.

Laryngeal spasm is defined as laryngeal obstruction due to partial or complete spasm of the intrinsic or extrinsic muscles of the larynx.[59] Two mechanisms are in effect: (1) Reflex closure of the glottis is due to a shutter-like adduction of the vocal cords, i.e., it involves the intrinsic laryngeal muscles. The resulting airway obstruction is incomplete and intermittent and occurs during inspiration or expiration. It usually occurs in response to somatic sensory stimulation, such as suctioning or the presence of an airway during light planes of anesthesia and during recovery. Treatment includes discontinuation of the stimulus and application of positive pressure with bag and mask, and administration of 100% oxygen. (2) Reflex closure of the larynx is due to a ball valve mechanism involving the extrinsic muscles, particularly the thyroid muscle[59] and occurs in response to visceral sensory stimulation and as an exaggerated response to stimulation of the superior laryngeal nerve.[114] It results in complete laryngeal obstruction. To treat the laryngeal spasm, the stimulus should be stopped or any irritant removed from the larynx such as secretions, blood, or a long airway. One hundred per cent oxygen should be administered. This spasm cannot be broken by positive pressure but may be terminated by severe hypoxia, hypercarbia, or deep anesthesia.[102] The incidence of laryngospasm is higher below the age of 9 years and even higher between 1 to 3 months of age.[91] It is commonest following extubation, in the presence of a nasogastric tube, and after oral endoscopies. The highest incidence was found in children with respiratory tract infections (95.8/1000).[91] Forcing the chin forward by strong pressure applied behind the angles of the jaw is often an effective maneuver to relieve the problem.[59] Attempts to force air into the pharynx under positive pressure will worsen the obstruction when the larynx is completely closed, since it leads to distention of the pyriform fossa, pressing the aryepiglottic folds more firmly against each other.[59] If forward movement in the temperomandibular joint does not relieve the airway obstruction, a small dose of succinylcholine, 0.5 to 1 mg/kg, together with atropine should be administered.[102] Respiration must then be supported by positive pressure ventilation (e.g., Ambu bag) until full muscle function has recovered.

In the absence of intravenous access, succinylcholine, 4 mg/kg, can be given intramuscularly to relieve the laryngospasm.[102] This has, however, been associated with the development of acute pulmonary edema.[22] If hypoxia and bradycardia are developing, intubation without relaxation should be performed to prevent further deterioration. Direct spraying of the cords with lidocaine, 1 mg/kg, will facilitate intubation by relaxing the larynx.[10] If the airway cannot be established, cricothyrotomy may be lifesaving.[102] Laryngospasm following extubation may be prevented by injection of lidocaine, 2 mg/kg, intravenously 1 minute before extubation[10] and delaying extubation until the child is completely awake and resumes a normal breathing pattern after a cough.[70] Laryngospasm should be considered a serious complication, necessitating aggressive therapy. In a recently published review series[91] 5 out of every 1000 patients who developed laryngospasm sustained a cardiac arrest.

Relief of acute upper airway obstruction of various etiologies may result in development of negative pressure pulmonary edema.[70, 119] The pathophysiologic basis for the development of this noncardiac form of pulmonary edema includes creation of large negative intrathoracic pressure gradients across the alveolar-capillary membrane, hypoxic pulmonary vasoconstriction, and increased transmural pressure in the heart and great vessels with increased left ventricular afterload.[70, 119] Therapy includes positive pressure ventilation with positive end expiratory pressure and if necessary, forced diuresis with furosemide.

Postextubation Croup

Laryngeal or rather subglottic edema leads to the clinical picture of postintubation croup.[66] Infants are more prone to postintubation complications for the following reasons (in order of decreasing importance): (1) The infant has a smaller larynx. In the adult, 1 mm of edema produces only slight hoarseness. In the infant the same amount of edema reduces the lumen by 75% and produces serious airway

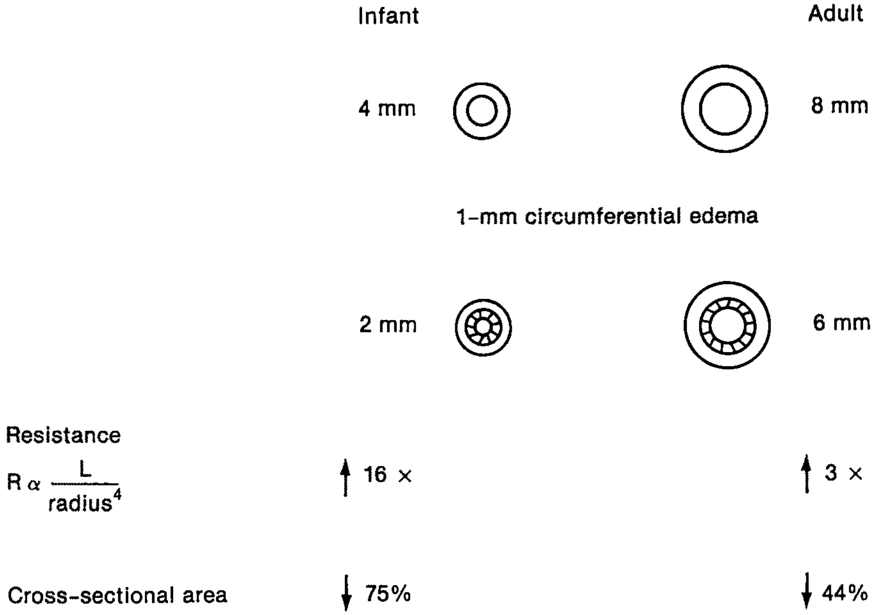

Figure 1. Relative effect of airway edema in infants and adults.

obstruction[38] (Fig. 1). (2) Loose areolar tissue is present in the submucosa of the subglottic area, where edema fluid easily accumulates. (3) The cricoid cartilage forms a complete ring.

The incidence of postextubation croup varies between 1% to 4% of intubated patients. It is most common in children 1 to 3 years of age.[66] Contributory factors include (1) tight-fitting endotracheal tube; (2) trauma during intubation; (3) long duration of intubation (greater than 48 hours); (4) movement of the head and, therefore, of the endotracheal tube during positive pressure ventilation; (5) surgery of the head and neck or bronchoscopy; and (6) hypotension.[66]

The incidence of postextubation croup is markedly diminished by use of an endotracheal tube that allows an initial audible leak at 25 to 35 cm H_2O.[33, 57]

Symptoms of postextubation croup usually occur within 30 to 60 minutes following extubation.[76] The diagnosis is made on the basis of stridor, thoracic retraction, hoarseness, croup-like cough, and varying degrees of respiratory obstruction.

Treatment includes (1) positioning the patient upright, (2) inhalation of cool humidified oxygen, and (3) inhalation of racemic epinephrine.[66, 76] The 2.25% solution is diluted with sterile water to a total volume of 2 mL in the following proportion: 1:8 $<$ 5 kg; 1:6 for

5 to 10 kg; 1:4 for 10 to 15 kg; and 1:3 for 10 to 20 kg.[21, 24] It may be delivered by face mask with nebulization or intermittent positive-pressure breathing.

If symptoms cannot be controlled with inhalation every 30 minutes or if hypoventilation with rising arterial Pco_2 occurs or the patient appears obtunded, reintubation is necessary to stabilize the airway.[74, 76] Reintubation should be performed with an endotracheal tube at least one size smaller than the original endotracheal tube and an auscultatory air leak should be detectable over the larynx at 25-to-30-mm water airway pressure to avoid long-term sequelae. The use of steroids for the treatment of postextubation croup remains controversial.[117] The drug and dosage most commonly recommended is dexamethasone, 0.5 to 1.0 mg/kg.[74, 76] Reintubation in croup is not associated with significant subglottic stenosis.[19]

Respiratory Depression

Respiratory depression in the recovery room may occur for a variety of reasons.

Ventilatory reflexes are depressed by anesthetics in a dose-dependent fashion. Although the response to Co_2 is significantly depressed above 1 MAC for halothane, enflurane, and isoflurane with flattening of the Co_2 response curve,[93a] the response to hypoxemia is already severely depressed at subanesthetic concentrations.[122] Patients who rely on their chemoreceptor drive for ventilatory control (e.g., pickwickian syndrome) may develop severe hypoventilation following anesthesia with potent inhalational anesthetics. Narcotics and ketamine produce depression of respiration with right shift of the Co_2 response curve.[9] Rate is primarily affected. Bolus doses of ketamine flatten the Co_2 response curve.[53] The newer synthetic opiates like fentanyl can cause chest wall rigidity and cause more marked ventilatory depression in the newborn and young infant compared with the older child.[65, 124] Morphine sulfate causes more profound respiratory depression in newborns in equi-analgesic doses when compared with meperidine, presumably because of higher blood brain permeability.[120]

Barbiturates depress rate and depth of respiration, which may be mitigated by noxious stimuli. After arrival in the postanesthesia care unit, following large doses of barbiturates, severe respiratory depression may occur, because the patient is now undisturbed.[121] Sedatives of the benzodiazepine group cause only mild respiratory depression due to a reduction in tidal volume.[121] However, in combination with narcotics, they may lead to severe depression of respiration and apnea.[110]

Due to the high minute ventilation to functional residual capacity ratio of infants and children, recovery from inhalational anesthetics is rapid. Although on a theoretical basis, recovery from the less soluble agents isoflurane and enflurane should be more rapid, clinically no difference is seen,[20] and airway irritability and stormy emergence is more common particularly with enflurane.[111]

Residual neuromuscular block, because of inadequate reversal, may lead to muscle fatigue (particularly of the diaphragm) and respiratory failure. This is of particular importance during the first months of life when the neuromuscular function is still immature and the diaphragm has a high proportion of type II (nonfatigue resistant) muscle fibers.

Reversal of neuromuscular blockade cannot be ascertained by voluntary clinical signs like head lift and grip strength in the infant. However, reflex leg lift appears to be an adequate substitute because it is associated with maximal negative inspiratory force of at least -32 cm H_2O.[77] This, and a crying vital capacity of greater than 15 mL/kg, have been shown to demonstrate adequate ventilatory reserve.[106] Use of a nerve stimulator to measure the response to supramaximal stimulation of, usually, the ulnar nerve, and using the train-of-four ratio of the force of contraction as assessment of degree of neuromuscular blockade[2] gives an objective measurement of reversal of neuromuscular block. A train-of-four ratio higher than 0.8 indicates adequate reversal.[38]

Pre-existing pulmonary disease and decreased ventilatory function as a result of surgery (e.g., cardiothoracic) may result in respiratory failure.[44]

Treatment of respiratory depression involves the following:

1. Stir-up regimen by the recovery room personnel stimulates spontaneous respiration and is often sufficient. This involves verbal and tactile stimulation.
2. Naloxone (Narcan) may be used to reverse narcotic-induced respiratory depression. It is a pure narcotic antagonist, and acts by competitive inhibition. Its duration of action is short-lived (30 to 60 minutes) and, hence, respiratory depression may recur. Thus, patients must be observed closely, or an intravenous dose of naloxone must be followed by intramuscular dose to provide a longer-lasting effect.[88]
3. Residual muscle relaxant may be reversed with an anticholinesterase-anticholinergic combination.
4. Controlled ventilation may be indicated to maintain a normal $Paco_2$ and to enhance the excretion of inhalational anesthetics by increasing alveolar ventilation.

Pulmonary Aspiration

Factors that make the infant more vulnerable to regurgitation and aspiration of gastric contents include the following[103]:

1. The resting intragastric pressure may be higher in infants than in adults because of (a) the relatively small size of the stomach; (b) excessive

air swallowing during crying; (c) encroachment of other abdominal organs; and (d) strenuous diaphragmatic breathing.

2. The infant has a shorter esophagus.
3. Relaxation of the gastroesophageal junction; regurgitation following feeding has been observed in normal infants up to 9 to 12 months of age. Gastroesophageal reflux has been well documented as a cause of apnea, wheezing, and pneumonia in small infants[12, 75] and laryngeal competence is decreased for 6 to 8 hours following extubation.[18]
4. The cough reflex may not be well developed.
5. Discordance of breathing and swallowing mechanisms may occur in premature and dyspneic infants.[103]
6. Atropine relaxes the tone of the esophageal-gastric junction.[92]

Aspiration continues to present a significant problem in pediatric anesthesia. Fifty per cent of aspirations occur in the postanesthetic period.[51, 115] Aspiration constituted 25% of all postanesthetic problems seen in the most recent series.[45] However, although aspiration caused 25% of anesthesia-related mortality in the 1960s,[51] perianesthetic mortality related to pulmonary aspiration has not been noted in recent reviews.[102a, 115]

Apnea and the Formerly Premature Infant

Apnea in the newborn and premature is defined as a respiratory pause of 20 seconds or longer, or a briefer episode of apnea associated with bradycardia, cyanosis, or pallor.[4, 101] Apnea of 5 to 10 seconds alternating with breathing is defined as periodic breathing.[101] Both are commonly seen in premature infants (25% to 60%). Etiology includes immaturity of respiratory control, episodic airway obstruction, alteration in sleep state, and diaphragmatic fatigue. The incidence of apnea episodes is increased with hypoxemia, sepsis, intracranial hemorrhage, patent ductus arteriosus, hyperthermia and hypothermia, and hypoglycemia and hypocalcemia.[101] Treatment of idiopathic apnea consists of therapy with theophylline.[29] Low-level CPAP also reduces the frequency of apneic episodes. Apnea is rarely seen in term infants. Apnea with bradycardia causes reduction in cerebral blood flow and may result in hypoxic-ischemic injury to the developing central nervous system.[94]

Since 1982,[112] it has been recognized that following minor operations (most commonly inguinal herniorraphy) infants who were born prematurely developed apnea episodes following an uneventful general anesthetic. Apnea occurred whether or not a previous history of apnea was present,[69, 72] but occurred more frequently and at a higher postconceptual age if a history of apnea or bronchopulmonary dysplasia was present. The majority of infants developing apnea or periodic breathing were less than 44 weeks postconceptual age and less than 3000-g body weight.[112, 79] Apneic episodes in all series occurred within 12 hours of surgery and were common if neuromuscular blocking agents had been used intraoperatively. Intraoperative therapy with caffeine, 8 to 10 mg/kg, prevented episodes of prolonged apnea in infants less than 44 weeks postconceptual age and with a history of apnea. No general consensus has been reached about the postconceptual age up to which in-hospital monitoring is required in former premature infants following minor surgery. Below 44 weeks postconceptual age, the incidence of postoperative apnea is highest. Because apnea has been observed even in a term infant, below this age[80] monitoring for apnea following anesthesia may be indicated. Infants with a history of apnea who are less than 48 weeks postconceptual age are at significant risk and require monitoring for at least 12 hours.

Acute Respiratory Failure

Acute respiratory failure is defined as an impairment of alveolar ventilation and pulmonary gas exchange resulting in failure to deliver sufficient oxygen to meet the demands of the body or inability to eliminate carbon dioxide adequately.[232] The impairment in gas exchange poses an immediate threat to life.

Causes for acute respiratory failure vary with the age of the patient. Outside the newborn period, respiratory failure is most commonly due to bronchopneumonia, upper airway obstruction, congenital heart disease, and asthma.[36, 118] Postoperatively, respiratory failure can occur either due to pre-existing lung disease, e.g., asthma, cystic fibrosis, upper respiratory tract infection, or as a complication of surgery (particularly thoracic or cardiac) or as a result of anesthetic overdose (respiratory depression from anesthetic agents or persistent neuromuscular blockade).

Clinical criteria for the diagnosis of respiratory failure in infants and children include the following: (1) severe inspiratory retractions and use of accessory muscles of respiration, flaring of alae nasi; (2) irregular respirations, tachybradypnea, and prolonged apnea; (3) diminished or absent breath sounds; (4) cyanosis in 60% oxygen; (5) decreased level of consciousness; (6) poor skeletal muscle tone; and (7) signs of cardiovascular collapse: absent or diminished pulse and blood pressure, excessive tachycardia pulsus paradoxus > 12 mm Hg.[27, 118]

Physiological criteria include[27, 118] hypercarbia with $Pa_{CO_2} \geq 65$ mm Hg; hypoxemia with $Pa_{O_2} \leq 70$ mm Hg in F_{IO_2} 0.6; for newborn, $Pa_{O_2} \leq 55$ mm Hg in F_{IO_2} 0.6. Three clinical and any one physiological criterium confirm diagnosis of acute respiratory failure.

Changes in pulmonary mechanics requiring mechanical ventilation include those listed in Table 1.[36]

Treatment aims at improvement of pulmonary gas exchange. Because loss of lung volume occurs with anesthesia resulting in increased shunting, a trial of CPAP may be used to improve oxygenation.[28, 63] CPAP may be applied by nasal prongs in the newborn and

Table 1. *Changes in Pulmonary Mechanics Indicating Need for Mechanical Ventilation*

Vital capacity, mL/kg	< 15
Forced expiratory volume at 1 second, mL/kg	< 10
Negative inspiratory force, mm H_2O	< 20

via face mask in the infant and child. CPAP delivered by face mask is generally poorly tolerated and leads to gastric distention.[27] CPAP is therefore best applied via endotracheal airway. CPAP results in an increase in functional residual capacity and alveolar and airway dimensions. The increased lung volume increases compliance and reduces the work of breathing. The optimal level of CPAP is defined as that level at which maximal oxygen transport is achieved (oxygen content × cardiac output).[113] Excessive levels of CPAP increase dead-space ventilation, which may lead to hypercarbia, reduction in venous return and cardiac output, and barotrauma of the lung.

If oxygenation cannot be improved or if hypoventilation and hypercarbia are present, mechanical ventilation via an artificial airway must be instituted.

Assisted ventilation is started with tidal volumes of 10 to 15 mL/kg and respiratory rates of 25 to 30 for the infant and 16 to 20 for the child. With pressure and time-flow preset, ventilator adequacy of chest expansion and breath sounds will guide initial setup, and analysis of arterial blood gases will determine final adjustment for each mode of ventilation. Positive end expiratory pressure (PEEP) is added in increments of 2 to 3 mm H_2O to improve oxygenation. Because the resistance of the upper airway is bypassed by endotracheal intubation, a physiological PEEP of 2 to 3 mm H_2O should be provided for all intubated children to minimize loss of functional residual capacity.[83] If a PEEP of higher than 10 mm H_2O is required to achieve adequate oxygenation, measurement of cardiac filling pressures should be instituted (central venous or pulmonary arterial pressure) to judge adequacy of cardiovascular performance. Ventilation with high mean intrathoracic pressures usually requires augmentation of cardiac filling.[26] With interstitial or alveolar disorders, commonly seen in the postanesthetic period, ventilation with large tidal volumes at relatively low ventilatory frequencies and with low inspiratory flow and end-expiratory plateau maximizes the distribution of gases with minimal peak airway pressure. Patients with obstructive airway disease (asthma, bronchiolitis) are better ventilated with higher inspiratory flow rates, shorter inspiratory times, and longer expiratory pauses to allow for exhalation from obstructed portions of the lung, thus minimizing the problem of air trapping and alveolar rupture.

Table 2. *Endotracheal Tube Sizes Below 2 Years of Age*

AGE	SIZE (*Internal Diameter in mm*)	LENGTH (*cm*)
Premature		
<1000 g	2.5	8–10
1000 g	3.0	10
2500 g		
Newborn to 6 months	3.0–3.5	10–11
6 months to 1 year	3.5–4.0	12
1–2 years	4.5	13

Provision of mechanical ventilation by positive pressure requires an artificial airway. Endotracheal intubation is presently preferred for management of ventilatory failure in infants and children even for prolonged periods of time and is associated with minimal morbidity.[11] Tracheostomy is reserved for patients with upper airway anomalies or chronic lung disease requiring prolonged ventilatory assistance and for the provision of home ventilator care.

Endotracheal Intubation in Children

Size. The optimal size is one that passes easily through the glottic and subglottic regions and produces a small leak of gas around the tube at 25 to 30 cm of water peak inspiratory pressure.[11] One formula that we use for the calculation of the appropriate size for an endotracheal tube follows:

$$\text{Internal diameter (mm)} = \frac{\text{age (years)}}{4} + 4$$

This formula is for uncuffed endotracheal tubes and is applicable for children over 2 years of age. Table 2 lists endotracheal tube sizes for patients younger than 2 years. A few patients will require an endotracheal tube that is smaller or larger.

Cuffed endotracheal tubes should not be used in children less than 7 to 8 years of age, because they reduce the size of the lumen, thereby increasing airway resistance and putting pressure on the delicate tracheal mucosa, thus increasing the incidence of subglottic damage. The correct size of a cuffed endotracheal tube is 0.5-mm internal diameter less than that calculated by the previously mentioned formula.

The composition of endotracheal tubes is important. Toxic substances form on gamma-ray–sterilized polyvinyl chloride (PVC) tubes after resterilization with ethylene oxide. The clear, polyvinyl, disposable tubes that have been tissue-implant tested (Z 70) are the most satisfactory ones for general use. Because they are easier to stabilize, nasotracheal tubes are preferred for long-term ventilatory management. Endotracheal tubes move with movement of the neck. Flexion moves the tip caudal, extension cephalad.[35, 122] A chest roentgenogram should be obtained after each endotracheal intubation to ascertain tube position and should be correlated with neck position.[116]

Complications of prolonged nasotracheal intubation include colonization with gram-negative organism, cosmetic nasal deformity, chronic ear infection, silent aspiration obstruction because of blockage or kinking, and accidental dislodgement. Partial obstruction results in a significant increase in airway resistance. Prevention of partial or complete obstruction requires adequate humidification of the inspired air and frequent suctioning, particularly in the presence of increased secretions. Since effective coughing is not possible with an artificial airway in place, chest physiotherapy should be used to propel mucous secretions centrally where they can be removed by suctioning. Suctioning should be performed with a sterile technique using sterile gloves and catheters after preoxygenation with 100% oxygen. The suction catheter should not completely occlude the lumen, since excessive subatmospheric pressure may result in pulmonary collapse. Endotracheal suctioning always results in loss of functional residual capacity and may cause hypoxemia.[52] Suction should only be applied on removal of the catheter. Sidehole catheters may result in suction damage to the walls of the trachea. Trauma from suction catheters may result in ulceration and stenosis of the distal trachea and main stem bronchi.

Weaning from ventilatory support can be attempted in the presence of a stable cardiovascular system and with the child awake and alert. The infant and child should be able to generate at least -20 mm H_2O negative inspiratory pressure and a vital capacity of 10 mL/kg. Blood gases should be stable with an F_{IO_2} of less than 0.5 and arterial P_{O_2} of more than 6 mm Hg. Pa_{CO_2} should be less than 60 mm Hg and PEEP less than 10 cm H_2O.[27, 83] The patient is weaned to a CPAP of 2 to 3 mm H_2O and after a period of spontaneous ventilation on CPAP of 2 to 3 cm H_2O without deterioration of blood gases or signs of respiratory fatigue or stress, extubation is carried out.

In the presence of unilateral lung disease, the good lung should be positioned *uppermost* to optimize gas exchange.[30]

CARDIOVASCULAR COMPLICATIONS

The cardiovascular system is not fully matured in the infant. The heart contains less contractile elements and cardiac output is rate dependent.[27] The sympathetic innervation is also incomplete, predisposing to exaggerated vagal responses. Cardiac output under basal conditions, however, is twice that of an adult because of the high energy demands. Because of the heart rate dependency of cardiac output, bradycardia is more worrisome than tachycardia. Episodic bradycardia, sinus bradycardia with nodal escape, and varying degrees of atrioventricular block, as well as atrial and ventricular extrasystoles can be observed in normal newborns and particular premature infants as a result of autonomic imbalance and developmental changes in the conduction system.[89] Bradycardia and apnea may follow vagal stimulation (e.g., suctioning).

Although the newborn heart is sensitive to vagal stimulation, it is relatively insensitive to the direct effects of hypoxemia. In adults cardiac output and myocardial contractility are decreased by hypoxemia, whereas in the newborn under similar conditions high-energy phosphate levels and myocardial function are maintained, presumably because of the newborn's ability to use anaerobic metabolic pathways more efficiently.[44] However, if hypoxemia is combined with acidosis, myocardial function declines.[46]

As a result of the immaturity of the sympathetic nervous system, cardiovascular reflexes that maintain perfusion under conditions of hypovolemia are not fully functional. The infant is unable to mount massive vasoconstriction to maintain cardiac output. Maintenance of an adequate blood volume and central venous pressure is therefore essential for maintenance of blood pressure and organ perfusion. Inhalational anesthetics depress myocardial function to various degrees[57, 59] and suppress baroreceptor response, an effect that may persist into the postanesthetic period.

During the first weeks of life the infant's circulation remains in a transitional state. The two main fetal channels diverting blood flow from the lungs in utero, the ductus arteriosus and the foramen ovale, are only functionally closed. The main stimulus to ductal closure is the postnatal increase in arterial oxygenation, which causes constriction of the ductal musculature.[89] The foramen ovale closes as left atrial pressure exceeds right atrial pressure following the increase in pulmonary blood flow and systemic vascular resistance after birth. Conditions that increase pulmonary vascular resistance, particularly hypoxia and acidosis, may lead to reopening of these fetal channels, bypassing the lung and leading to severe cyanosis and rapid cardiovascular deterioration.[49, 93]

As pointed out previously, the immaturity of vascular reflexes requires maintenance of an adequate filling pressure to maintain cardiac output, blood pressure, and organ perfusion. Blood pressure in the absence of compensatory vasoconstriction becomes an indicator for adequacy of blood volume and can be used as a guide for volume replacement.[89] Table 3 lists normal values for heart rate and blood pressure for various ages. Blood pressure should be measured with a

Table 3. *Normal Values for Blood Pressure and Heart Rate*

	HEART RATE		BLOOD PRESSURE	
	Mean	*Range*	*Mean Systolic* ±2 *SD*	*Mean Diastolic* ±2 *SD*
Newborn	145	70–190	65 ± 18	40 ± 16
1–11 months	120	80–160	96 ± 27	65 ± 27
2 years	110	80–130	95 ± 25	61 ± 24
4 years	100	80–120	99 ± 20	65 ± 20
6 years	100	75–115	95 ± 15	55 ± 9
8 years	90	70–110	100 ± 16	55 ± 9
10 years	90	70–110	110 ± 17	58 ± 10
12 years	88	70–110	115 ± 19	59 ± 10
14 years	83	60–105	118 ± 19	60 ± 10

cuff the width of which is equivalent to 40 to 50% of the circumference of the extremity.[63, 75]

Blood volume in the newborn and infant is larger on a weight basis than later in life. In the newborn, blood volume is 9 to 10% of body weight and declines to adult values (7% of body weight) by 2 years of age.[89, 108] In the newborn, the expanded blood volume consists mostly of red cell volume. The more immature the infant the larger the red cell volume per unit body weight.

Water is the main constituent of body mass, the remainder consisting of fat, skeletal minerals, and cell solids.

Total body water can be divided into intracellular and extracellular fluid space. Total body water declines from 78% of body weight at birth to 65% at 1 year of age and 55 to 60% in the adult. Extracellular water makes up a much larger proportion of total body water in the infant. A small premature may have an extracellular fluid space of 60% of total body water. At birth, extracellular water constitutes 45% of total body water, which rapidly declines in the first few months. It reaches 25 to 30% by 1 year and adult levels of 20% at puberty (Fig. 2).

Renal function at birth is immature. Glomerular filtration rate (GFR) is only 25% of adult values.[42] This results in an inability to excrete large fluid loads rapidly. The concentrating capacity is also poorly developed as is the capacity for secretion and reabsorption in the tubules. As a result, the immature kidney is unable to conserve either water or salt, and fluid deprivation, abnormal losses, or hydration with nonsodium containing fluids can rapidly lead to severe dehydration and electrolyte imbalance, particularly hyponatremia.[43] Full maturation of renal function is not achieved until the end of the first year of life. Under baseline conditions normal adults have an oral water requirement of approximately 35 mL/kg/day, and normal infants, 130 to 150 mL/kg/day. This explains their greater propensity for dehydration. Besides water, electrolytes and calories have to be provided

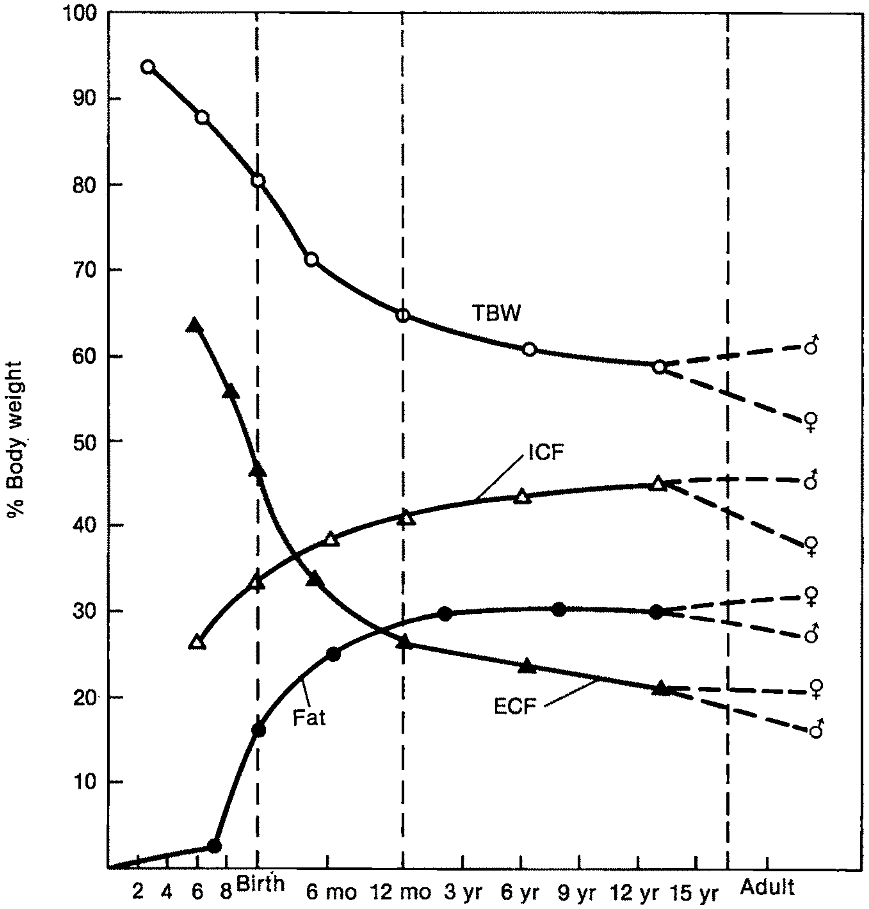

Figure 2. Body water compartments. Changes with age. Abbreviations: TBW-total body water; ICF-intracellular fluid; ECF, extracellular fluid. (*Modified from* Friis-Hansen B: Body water compartments in children. Pediatrics 28:169–181, 1981; and Winters RW: Regulation of normal water and electrolyte metabolism. *In* Winters RW (ed): The Body Fluids in Pediatrics. Boston, Little Brown, 1973, pp 95–112.)

to maintain a patient in zero balance.[21] In the perioperative period, maintenance fluid therapy generally only addresses water and electrolyte balance and caloric requirements only insofar as to prevent ketosis and gluconeogenesis. The latter can be provided by infusion of 5% dextrose solution. Fluid and electrolyte requirements are conveniently based on caloric need and body weight.[21]

Usual requirements for water, electrolytes, and glucose are summarized in Table 4. These requirements are met by 5% dextrose with 0.25% NaCl and 20 mEq K^+/L in infants. Newborns and premature infants require glucose concentration to be increased to 10% to prevent hypoglycemia. The sodium concentration of maintenance solu-

Table 4. *Maintenance Requirements for Water, Electrolytes, and Glucose*

	PER 100 CALORIES
Water	100 mL
Sodium	2.5 mEq
Potassium	2.5 mEq
Chloride	5.0 mEq
Glucose	5 g

tions needs to be increased to 40 mEq/L in premature infants to prevent hyponatremia. The amount of fluids required based on caloric need varies with weight and is outlined in Table 5. Glucose-containing solutions should not be given to replace abnormal losses and should not exceed 15 to 20 mL/kg/hour to avoid hyperglycemia and hypersmolarity, resulting in cellular dehydration and increased renal water losses.[21] In premature infants hyperglycemia may cause intracerebral hemorrhage.[64]

Maintenance fluid requirements are increased by fever (12% for each degree above 37°C.)[124] They are also increased by hyperventilation and hypermetabolic states, e.g., thyrotoxicosis and decreased during hypothermia without compensatory increase in metabolic rate (12% for each degree below 37°C.)[124]

REPLACEMENT FLUID THERAPY

The purpose of replacement fluids as opposed to maintenance fluids is to correct body fluid deficits caused by external losses (vomiting, nasogastric suction) or internal sequestration (burns, loss into bowel wall and lumen in intestinal obstruction or due to surgery). Surgery results in less sodium and water retention and less catabolic response in the infant when compared with the adult.[73] However, there appears to be increased renal potassium loss. Translocation of isotonic fluid into a nonfunctional interstitial compartment is related to the degree of surgical trauma.[56] It is highest with intra-abdominal procedures. Unreplaced translocation, i.e., third-space losses, results in clinical signs of dehydration.

Table 5. *Maintenance Fluid Requirements*

0–10 kg	100 mL/kg/24 hours
11–20 kg	50 mL/kg/24 hours for each kg over 10
over 20 kg	20 mL/kg/24 hours for each kg over 20

Table 6. *Clinical Signs of Dehydration*

	MILD	MODERATE	SEVERE
Body weight	−5%	−10%	−15%
Skin turgor	↓	↓ ↓	↓ ↓ ↓
Mucous membranes	Dry	Very dry	Parched
Skin color	Pale	Gray	Mottled
Urine	Mild oliguria	Oliguria	Severe oliguria and acotemia
Blood pressure	Normal	Normal	Reduced
Pulse	±	↑	↑ ↑

Third-space fluid is derived initially from the plasma volume, which in turn is replenished from the interstitial fluid. Thus, this fluid loss is nearly isotonic and suitable replacement fluids include lactated Ringer's solution, normal saline, and Normosol. Large amounts of normal saline may cause hyperchloremic acidosis owing to the inappropriate high chloride content (150 mEq/L in 0.9% NaCl versus 103 mEq/L in serum).

The amount of third-space loss and, therefore, the amount of replacement required, depends on the severity of trauma. For example, for minimal surgical trauma (hernia repair) 1 to 2 mL/kg/hour is required; for moderate surgical trauma (pyloromyotomy) 2 to 4 mL/kg/hour; and for major surgical trauma (bowel resection) 4 to 6 mL/kg/hour.

Much larger amounts may be required to achieve the endpoint of adequate replacement therapy, i.e., a stable blood pressure and adequate urine output.[21] A minimal urine output of 0.5 to 1 mL/kg/hour should be maintained under any circumstances.[73]

In premature and small infants requiring large amounts of replacement fluids, one fourth to one third should be replaced with 5% albumin to effect a more equal distribution between extracellular and intracellular fluid and plasma volume.[85] Ongoing abnormal losses owing to drains should be measured and replaced with appropriate solutions. Clinical signs of dehydration are outlined in Table 6.[31] Hypotonic dehydration generally results in more severe signs of dehydration; hypertonic dehydration in less. Table 7 lists the composition of replacement fluid for specific gastrointestinal losses.[124]

BLOOD REPLACEMENT

The allowable whole blood loss in pediatric surgical patients is calculated to attain a final postoperative hematocrit of not less than 30% for infant and children and 40% for prematures and newborns.[58]

Table 7. *Replacement Solutions for Gastrointestinal Losses*

	Na^+ mEq/L	K^+ mEq/L	Cl^+ mEq/L	HCO_3 mEq/L	DEXTROSE g/L
To replace gastric drainage	140	15	155	0	0
To replace small intestine losses	140	15	115	40	0
To replace diarrheal losses	40	40	40	40	50.0

Modified from Winters RW: Maintenance fluid therapy. *In* Winters RW (ed): The Body Fluids in Pediatrics. Boston, Little Brown and Co, 1973, pp 113–133.

Estimated blood volume (EBV) = weight in kg × y mL/kg, where y = 90 for newborn, 80 up to 1 year, 70 up to 12 years, and 65 for adolescents.

$$\text{ERCM} = \text{EBV} \times \frac{\text{Hct}}{100} \qquad \text{(A)}$$

$$\text{ECRM}_{30} = \text{EBV} \times \frac{30}{100} \qquad \text{(B)}$$

$$\text{ARCL} = \text{A} - \text{B}$$

$$\text{AWBL} = (\text{A} - \text{B}) \times 3$$

where ERCM = estimated red cell mass (depends on patients preoperative hematocrit); ERCM_{30} = estimated red cell mass at a hematocrit of 30%; ARCL = allowable red cell loss; and AWBL = allowable whole blood loss.

The assumption made is that blood loss is occurring at a hematocrit of 30%. For practical purposes the management protocol presented in Table 8 may be used.

When replacing blood loss with isotonic electrolyte solution, each milliliter of blood has to be replaced with 3 mL of crystalloid, because the latter is also redistributed to the interstitial fluid volume.[127] Postoperative anemia can be treated with a transfusion of packed red cells, which have an average hematocrit of 75. One mL/kg of packed cells increases the hematocrit by 1 to 1.5%.[17, 50a]

Table 8. *Management Protocol*

AMOUNT OF BLOOD LOSS	THERAPY
Blood loss is less than one third of allowable whole blood loss	Ringer's lactated solution
Blood loss is more than one third but less than total allowable whole blood loss	Colloid, preferably 5% albumin
Blood loss is equal to or more than allowable whole blood loss	Blood

Persistent surgical bleeding in the postanesthetic period may indicate presence of a coagulopathy, particularly after massive intraoperative blood transfusion. In newborns and particularly premature infants, sepsis may cause depression in platelet count.[58] Clinical bleeding may be apparent with counts below 50,000. Production of various coagulation factors is reduced in the neonate and infant. Factors II, X, VII, IX, and XII are affected, and adult levels are sometimes not reached until 12 months of age. For this reason, prothrombin (PT) and partial thromboplastin (PTT) times vary widely in this age group. PT may range from 13 to 20 seconds and PTT from 45 to 70 seconds.[58] Treatment with specific blood components or factors may be indicated.[17] Transfusion of 0.3 U of platelets per kg will usually result in a platelet count above 65,000/mm^3.[89] Replacement of coagulation factors in the form of fresh frozen plasma may be required in patients after the loss of one blood volume, particularly if blood replacement is carried out with packed or frozen red cells. Replacement is calculated as 20 to 30% of blood volume lost as volume of fresh frozen plasma (FFP).[24] Our practice is to transfuse equal volumes of packed red cells and fresh frozen plasma after one blood volume loss. Since fresh frozen plasma has the highest citrate content of any blood product, rapid administration of this component may result in a significant fall of ionized calcium, particularly in the small infant or newborn with reduced calcium stores. If the rate of administration of FFP exceeds 1 mL/kg/minute, clinical signs of hypocalcemia may occur, particularly cardiovascular depression.[24] Therapy with intravenous calcium during rapid infusion of FFP is required to avoid complications. Calcium chloride, 2.5 mg/kg, or calcium gluconate, 7.5 mg/kg, are equally efficacious.[25]

NAUSEA AND VOMITING

Vomiting is a frequent and unpleasant sequel of general anesthetics in children. It is more common after certain procedures, particularly strabismus repair in which it occurs in 41 to 85% of patients.[55, 71] Vomiting is more common in the presence of surgical pain. Severe vomiting may preclude patients discharge after a planned outpatient surgery, result in dehydration, or in the early stages of recovery, may lead to aspiration and development of aspiration pneumonitis. Unconscious patients should be positioned on their sides to prevent aspiration if vomiting should occur. Intraoperative pretreatment with droperidol, 0.075 mg/kg intravenously, significantly lowers the incidence of postoperative vomiting. For greatest effectiveness it should be given 10 minutes before manipulation of the eye.[71] Droperidol may be given in the recovery room in case of vomiting but is less effective under these circumstances. Treatment with droperidol does not cause delay in discharge but appears to provide a smooth recovery without restlessness or agitation. Oral pretreatment with an equal dose of droperidol is less effective in the prevention of vomiting.[90]

TEMPERATURE REGULATION

Maintenance of normal body temperature is of major importance in caring for pediatric surgical patients. Body temperature is the result of a balance between heat production from metabolism and heat loss due to environmental factors. Heat is produced by activity, metabolism of food, particularly proteins (specific dynamic action), and in response to lower environmental temperatures.[67] In newborn infants, a major source of metabolic heat production rests in brown fat located between the scapula, the vessels of the neck, in the axilla and the mediastinum, and around the kidneys and adrenals. This tissue is particularly rich in mitochondria. Cold exposure or norepinephrine infusion will result in large increases in fat metabolism in these tissues with concomitant heat production, the so-called nonshivering thermogenesis. The maximal metabolic rate of brown fat is approximately 40 times the whole body metabolic rate with an oxygen consumption of 600 mL/kg/minute.[68] Hypoxia prevents heat production in brown fat. The sedated infant is unable to increase heat production by activity. Involuntary muscle activity, i.e., shivering, is practically absent under 3 to 6 months of age.[70]

Heat is distributed predominantly by convection through the circulation. Thermal insulation is minimal in the small infant with poorly developed subcutaneous fat and high skin blood flow. The disproportional large head surface area combined with a high cerebral blood flow contribute to the major influence of this area on temperature maintenance and whole body metabolic rate.[67] Heat loss from the organism occurs predominantly through radiation and convection. Radiation of heat occurs to the nearest surface, regardless of the temperature of the surrounding air and is proportional to the temperature difference between the radiating body and the surface. Convective heat loss is due to air movement around the organism. Radiation and convective heat loss are most efficiently reduced by reduction of exposed body surface by the use of blankets, caps, and wrapping in plastic foil. Conductive heat loss to air occurs and is negligible because of the low thermal conductivity of air. Considerable amounts of heat, however, can be lost to cold mattresses or blankets. Heat loss due to evaporation occurs from the skin and respiratory tract. This heat loss varies with ambient humidity, and, for example, the presence of wet skin. The increased alveolar ventilation, the large body surface

Table 9. *Recommended Environmental Temperatures for Various Ages*

Premature or neonate	27°C
Infant, 1–6 months	26°C
6 months to 2 years	25°C

area relative to weight, and the more water-permeable skin of the neonate lead to higher evaporation water losses in this age group. In the term baby, 20 to 30% of metabolic heat is lost in this manner.[67] A thermoneutral state is achieved when vasomotor adjustments alone without changes in metabolic rate are sufficient to maintain normothermia.[67] The set point for temperature in a neutral thermal environment is a rectal or core temperature of 36.5 to 37°C. The sensors for thermoregulation are predominantly located in the skin, particularly of the face and abdomen, and their sensitivity changes with the age and weight of the infant. In small infants a gradient of only 1.5°C between skin and environment or rectal and environmental temperature may be permissible for minimal O_2 consumption. In older infants a gradient of 2.5 to 5°C is generally acceptable. A skin temperature of the abdominal wall of 36.5°C, corresponding to an average skin temperature of 35 to 36°C is usually optimal.

As mentioned previously, reduction of exposed body surface area significantly reduces cold stress. Exposure to lower than thermoneutral environmental temperatures has multiple deleterious consequences. Nonprotein energy stores are depleted, release of catecholamines with increase in pulmonary vascular resistance occurs followed by hemorrhage and death.[67] To avoid the metabolic stress of temperature maintenance, not only core but also skin temperature has to be maintained. Stimulation of temperature-sensitive receptors located particularly in the face by, e.g., cold drafts (high flow oxygen), will result in a noticeable rise in metabolism.[76] Recommended environmental temperatures for various ages are listed in Table 9. The use of overhead infrared lamps can mitigate the effects of cool environment to a large extent.

Intensive efforts at prevention of heat loss will occasionally result in iatrogenic increases in temperature. Infants lose heat through peripheral vasodilatation and increase in evaporative heat loss (sweating). Premature infants are unable to sweat and the ability to sweat is not fully matured at birth. Dehydration and infection as well as excessive atropinization may cause elevation in body temperature. Metabolic rate is increased by 12% for each degree centigrade above 37°C and fluid therapy has to be adjusted accordingly. Physical measures to reduce elevated body temperature include convective heat loss with wet sponging and increased air circulation (fan) and the application of cold packs. An unusual cause for severe elevation in body temperature in the postanesthetic period with a concomitant maximal increase in metabolic rate is the development of the malignant hyperthermia syndrome.

PSYCHOLOGICAL PROBLEMS

The child characteristically has a lack of self-control, and it is usually difficult to persuade children of preschool age to act against their will. Hospitalization may lead to lasting psychological disturbances.[108] Induction of anesthesia and the waking-up process in the recovery room are critical times.[116] Although the infant less than 1 year old recovering from surgery is often satisfied with a loving hug to make him comfortable, the toddler waking up in strange surroundings requires comforting and compassionate care by the postanesthesia care team to avoid lasting psychological sequelae. Causes of hypoxia or hypoventilation should be excluded before sedating an agitated child. Protection from self-inflicted injury during a period of disorientation is important. The presence of a parent may be the best sedative for the child and reduce the incidence of postoperative psychological sequelae.[34]

LEVEL OF CONSCIOUSNESS: DELAYED AWAKENING

Delayed awakening may have multiple etiologies. The patient may have received large amounts of barbiturates or other sedatives and narcotics, and inhalational anesthetics may have been discontinued late and with the patient still hypoventilating; removal of these agents from the CNS will be delayed. Hypoxia and hypercarbia need to be excluded. Hypovolemia and hypotension with poor cerebral perfusion may result in drowsiness. Parenteral fluid therapy with solutions containing insufficient amounts of sodium may cause hyponatremia with CNS depression. In the small infant hypoglycemia may be present resulting in obtundedness. Residual neuromuscular block may lead to hypoventilation with hypercarbia. Hypothermia results in delayed awakening. Rare causes of delayed awakening include CNS pathology like intracranial hemorrhage, or the child may have sustained an intraoperative hypoxic-ischemic insult to the brain. Awakening with agitation and confusion is not infrequently seen in young children, particularly after inhalational anesthetics.[37] It is more common after the use of large doses of belladonna alkaloids, particularly scopolamine, and may be reversed under these circumstances with intravenous physostigmine at a dose twice that of the initial belladonna dose. Hypoxia may lead to confusion and should be excluded before treating restlessness. The use of ketamine (particularly if used in large doses) leads to delayed awakening with hallucinations. This is more common in older children. However, one of the most common reasons for agitation is postoperative pain. Relief of postoperative pain can be achieved by titrated doses of narcotics or prevented by judicious use of local and regional blocks.

POSTOPERATIVE PAIN RELIEF

Postoperative pain is a common problem in pediatric postanesthesia care. However, it has only recently been addressed.[14, 78] It appears that children frequently are provided with insufficient pain relief following surgery, partially because they may be unable to communicate. Signs of pain in infants and children include agitation, hypertension, hypoventilation with hypercarbia due to splinting, and hypoxemia due to atelectasis.

In preterm babies and small infants, reaction to pain causes release of stress hormones and results in a variety of cardiorespiratory problems. Bradycardia, poor circulation, metabolic acidosis, and increased ventilatory requirements have been described.[5]

Pain varies with anxiety and apprehension and can be reduced by adequate preoperative preparation. It is influenced by the site of surgery, perineal and orthopedic procedures being more painful than abdominal or thoracic ones.[109]

Anesthetic technique influences the severity of postoperative pain. Inhalational anesthetics have no analgesic properties. If no narcotics have been administered preoperatively or intraoperatively, early use of narcotics in the postanesthesia period may be required for pain relief and reduction of agitation. Pain is more easily prevented than treated. Small intravenous doses of morphine, 0.05 to 0.1 mg/kg, or fentanyl, 5 to 10 μg/kg, during emergence from inhalation anesthesia reduce pain and agitation on awakening. Continuous infusion with morphine, 0.02 mg/kg/hour, can be used in children over 6 months of age to provide postoperative analgesia.[16] Patient-controlled analgesia can be used for postoperative pain relief in children over 6 years of age.[99] Doses of morphine vary between 0.015 and 0.03 mg/kg/hour after a loading dose of 0.05 mg/kg, titrated with further doses to achieve pain relief, with a 4-hour limit set at 0.25 mg/kg. Low-dose ketamine, 0.5 to 1 mg/kg, following an inhalational anesthetic also provides excellent pain relief without cardiorespiratory depression or psychological side effects.[93]

Regional Anesthesia for Postoperative Pain Relief

The use of regional anesthesia or nerve blocks of the surgical area prior to emergence from general anesthesia provides excellent pain relief with minimal psychological alterations. If the block has been established after induction but prior to the surgical procedure, lighter levels of general anesthesia are frequently tolerated and recovery is therefore more rapid. In addition, because of the lack of surgical pain, recovery tends to be smoother.[15, 105] Patients require less narcotic and non-narcotic analgesics postoperatively and are able to ambulate easier, resulting in earlier discharge from an ambulatory setting.[54]

Distribution and metabolism of local anesthetics varies in the pediatric age group. Neonates and small infants have low concentrations of plasma proteins, particularly alpha-glycoprotein, which is responsible for plasma binding of lidocaine. Plasma concentrations of 2.5 μg/L are toxic in the neonate whereas adults rarely show signs of neurotoxicity below levels of 5 μg/L.[67] Plasma protein binding does not reach adult levels until after the first year of life. The low plasma cholinesterase levels seen in the newborn can result in delayed metabolism of ester-type local anesthetics like procaine if given in large doses. This may be of particular importance if red-cell cholinesterase is also low, as is seen during physiologic anemia at 2 to 3 months of age.[127] Amide-type local anesthetics cannot be metabolized well because microsomal enzyme systems of the liver are immature in the neonate and as much as 90% of the drug may be excreted unchanged in the urine.[84] After the first few months of life children eliminate drugs at a greater rate than adults, possibly because the liver mass constitutes a greater percentage of body weight and has relatively more metabolic sites for breakdown of local anesthetic drugs.[67] This accounts for the low blood concentrations seen after relatively large doses of local anesthetics in the older child. Plasma concentrations rise more rapidly to peak blood concentrations in children,[39, 49] which may be related to their greater cardiac output. However, elimination half-life is prolonged, which is thought to be due to the larger volume of distribution in children, which is related to the differences in body composition in the child.

After 1 year of age children are quite tolerant to high doses of local anesthetics and rarely exhibit signs of toxicity.[67] The dosage for local anesthetics is generally given as the maximum allowable dose. The addition of epinephrine retards absorption and permits the use of larger doses of local anesthetics. Intravascular injection or injection into a very vascular area can lead to signs of toxicity with very small doses. Table 10 lists anatomic sites and rapidity of absorption of local anesthetics in declining order. Table 11 lists maximum allowable amounts of local anesthetics at various sites.[97, 125]

Contraindications to the use of local anesthetic techniques are the same as in adults: infection at the site of injection or a systemic coagulopathy. Relative contraindications are the presence of anatomic abnormalities or degenerative nerve disease. Because most techniques are performed with the patient asleep or heavily sedated, a nerve stimulator should be used for localization in peripheral nerve blocks.

Table 10. *Speed of Absorption of Local Anesthetics from Various Anatomic Sites (in Declining Order)*

Fastest	Intratracheal
	Intercostal
	Caudal epidural
	Brachial plexus
	Distal peripheral nerves
Slowest	Subcutaneous infiltration

The most frequently performed blocks in pediatric patients are dorsal penile block for relief of circumcision pain, ilio inguinal-ilio hypogastric nerve block for inguinal herniorrhaphy, and caudal epidural for relief of pain associated with major genitourinary and lower-extremity orthopedic procedures. The simplest method for relief of postcircumcision pain is application of topical lidocaine to the incision. A block of the femoral nerve relieves the pain of fractures of the femoral shaft. Thoracotomy pain is relieved by blocking the first six intercostal nerves, whereas blocking of the six lower intercostals provides analgesia of the adbominal wall. For long-lasting postoperative pain relief local anesthetics may be injected repeatedly or by continuous infusions through the catheter placed epidurally by the sacral or epidural route[32, 41] or an interpleurally placed catheter positioned either by the surgeon or percutaneously.[81, 82] For caudal and epidural blockade in children, various dosage schedules have been proposed. A dose of 0.1 mL per segment per year of age of 0.25% bupivacaine or 1% lidocaine appears effective in prepubertal children.[104] Another formula to calculate dose is milliliter per segment = 0.056 × weight (kg).[114] The simplest method is to use 0.5 mL/kg of the desired local anesthetic solution for lumbar block, 1 mL/kg to achieve a lower thoracic level, and 1.25 mL/kg for a midthoracic level of analgesia.[5a] The

Table 11. *Local Anesthetic Agents and Maximal Dosage in Children*

DRUG (mg/kg)	SPINAL	CAUDAL	PERIPHERAL
Tetracaine	0.2–0.6 (minimal effective dose 1.5 mg)	2	2
Lidocaine	1–2.5	5	5
	With 1:200,000 epinephrine	7–10	7–10
Bupivacaine	0.3–0.4	3	3
	With 1:200,000 epinephrine	3–5	3–5
2-Chloroprocaine		15	15

total milligram per kilogram dose should be checked to be within the acceptable safe range of the drug.

The use of local anesthetics for pain control generally results in sympathetic blockade and motor weakness. Intrathecal or epidural narcotics result in similar pain relief and generally fewer side effects. Their use has not been widespread in pediatric anesthesia, mostly because patients require close observation for late respiratory depression for at least 12 hours following the injection of morphine.[68] Even in the absence of clinical signs of respiratory depression (decreased rate, increased end-tidal Co_2), the ventilatory response to Co_2 is depressed for up to 22 hours.[87] Preservative-free morphine sulfate may be injected epidurally or caudally at a dose of 0.1 mg/kg for postoperative pain relief for surgical procedures involving the legs, abdomen, and chest. Caudal epidural morphine has been used for pain control in children following open heart surgery[100]; however, pain relief was not complete and had to be enforced with small doses of parenteral narcotics. Side effects in children as well as in adults included (besides the rate occurrence of respiratory depression) pruritus, nausea and vomiting, and urinary retention. Pain relief following thoracotomies is of particular importance in pediatric patients with their limited ventilatory reserve. Pain may lead to instability of the chest wall, which must be stabilized by active muscle contraction and this, combined with diaphragmatic dysfunction, results in breathing at low tidal volumes, worsening airway closure, and leading to atelectasis. Pain relief allows the patient to cough and re-expand the lung, re-establishing adequate functional residual capacity. The undesirable side effects of epidural morphine, like nausea and pruritus and particularly, respiratory depression, may be reversed with small doses of naloxon intravenously without reserving analgesia.[68]

SUMMARY

The postanesthesia care of the pediatric patient covers the transition from general anesthesia with CNS depression and respiratory and cardiovascular instability to a state of full alertness with recovery of all protective reflexes. Familiarity with the specific requirements of infants and children and constant vigilance to prevent hypoxia and hypoperfusion allows safe recovery. In addition, plans to minimize psychological trauma due to separation or pain allow for a minimally disturbing hospital experience.

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The Neurosurgical Patient in the Postanesthesia Care Unit

*Irene Osborn, MD**

The postoperative care of the patient who has had neurosurgery involves attention to every organ system and its interaction with the central nervous system (CNS). Many of the special intraoperative considerations continue to apply in the postanesthetic period. Monitoring by the anesthesiologist must extend through transport into the postanesthesia care unit (PACU), as alterations in vital signs may signal the development of an untoward neurologic event.

In the PACU, patient vital signs are promptly measured. A basic neurologic assessment consists of a determination of the level of consciousness, degree of motor activity, and pupillary size, equality, and reactivity to light. The Glasgow Coma Scale was initially devised as a prognostic indicator after head injuries: it also has been used to assess neurologic status in postoperative patients[19] (Table 1).

THE CARDIOVASCULAR SYSTEM

The cardiovascular system can be profoundly affected by the CNS, and in turn can cause further compromise of an impaired CNS. Hypertension is the most frequently encountered problem in the postoperative period and is to be avoided in neurosurgical patients especially.

Autoregulation of cerebral blood flow is effective up to a mean arterial pressure of 150 mm Hg in the intact brain and is further limited in the compromised brain. Thus, a significant rise in systemic blood pressure can precipitate an increase in intracranial pressure. A period of uncontrolled hypertension may increase the likelihood of hemor-

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Table 1. *The Glasgow Coma Scale*

	RESPONSE	SCORE
Eyes open	Spontaneously	4
	To command	3
	To pain	2
	No response	1
Best motor response		
To command	Obeys	6
To pain	Localizes	5
	Flexion withdrawal	4
	Decorticate response (abnormal flexion)	3
	Decerebrate rigidity	2
	No response	1
Best verbal response	Oriented	5
	Disoriented	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1

Allowance should be made if the eyes are swollen shut or the trachea is intubated.

rhage at the operative site and can increase edema formation, particularly with an injured blood–brain barrier. Also, profound hypertension can provoke cardiac failure or myocardial infarction in patients with pre-existing disease[3] (Fig. 1).

Postoperative hypertension may be caused by vasoconstriction from hypothermia, which is exacerbated by shivering. Although many intracranial procedures require the use of diuretics intraoperatively, cerebrovascular surgery often dictates that patients maintain an adequate (or better) intravascular volume. This can lead to marked hypertension as vascular tone returns postoperatively. Hypoxia and hypercarbia are not to be overlooked as causes of hypertension by increasing catecholamine release. If nitroprusside was used intraoperatively for induced hypotension, a rebound effect may be seen, possibly due to interference with the renin–angiotensin system.[16] Use of naloxone or low-molecular weight dextran can contribute to increased blood pressure.[2]

Because of the serious consequences of hypertension, it should be treated promptly. However, one origin of hypertension, the Cushing reflex, is a protective mechanism to improve cerebral perfusion. Therefore, identifying the cause of hypertension is important prior to treatment. If hypercarbia and hypoxia have been ruled out, the administration of labetolol (5 to 10 mg) intravenously often provides rapid and effective treatment. One dose may be all that is required for transient blood pressure elevation. Persistent hypertension may be treated with additional bolus doses or an infusion of labetolol at

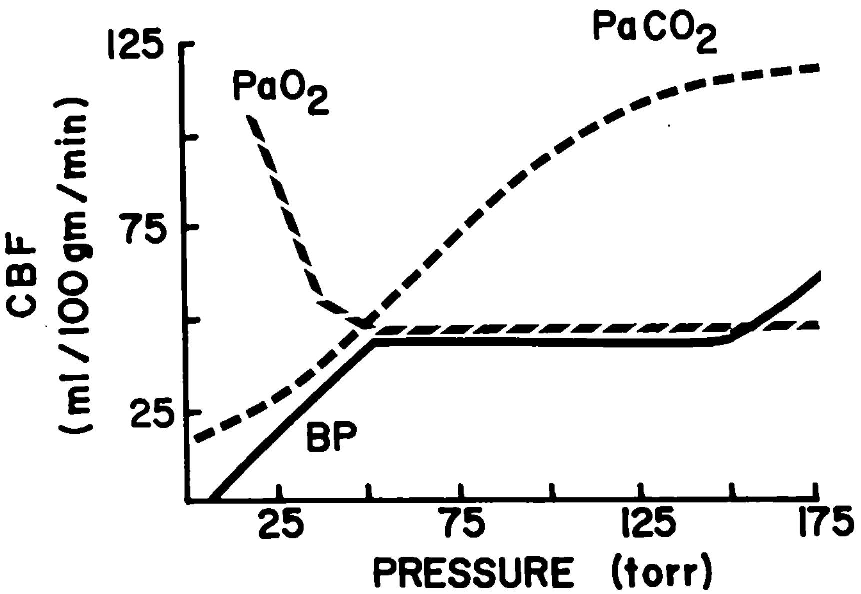

Figure 1. Schematic illustration of factors determining cerebral blood flow (CBF). Autoregulation is demonstrated by the maintenance of CBF over a wide range of mean arterial pressures. The response of CBF to changes in $Paco_2$ and Pao_2 is also represented.

2 mg/minute.[14] Recent studies have shown Esmolol to be as effective with more rapid onset of effects and a shorter half-life.[15]

Potent vasodilating agents may be hazardous if intracranial pressure is known to be elevated. Hydralazine (10 to 15 mg) may be administered if one has time to wait for its onset of effects (15 to 20 minutes). Sodium nitroprusside may be used following cerebrovascular procedures for *very* rapid control of hypertension. It may then be slowly decreased as other agents take effect.

Hypotension is seen less frequently but should be treated promptly as it may decrease cerebral perfusion. Decreased blood pressure may be caused by intraoperative fluid restriction or diuretic administration. Other causes include adrenal insufficiency, allergic reaction to antibiotics, drug interaction, myocardial infarction, or pump failure.[16]

An accurate assessment should be made of fluid intake and output, urine osmolarity, central venous pressure or pulmonary capillary wedge pressure if available. Unless the patient is profoundly hypotensive (and symptomatic) the head should remain elevated; the legs may be raised to improve venous return. Isotonic crystalloid solution or albumin should be administered at this time. If the patient is anemic, then blood or packed cells should be given.

Cardiac arrhythmias can be caused by CNS disease, metabolic abnormalities, or concurrent cardiac disease. Electrocardiographic changes such as ST depression of T wave inversion may occur, particularly after ruptured cerebral aneurysm or head trauma.[7] Increased serum catecholamine levels causing myocardial necrosis, central autonomic stimulation, or vascular spasm have been cited.[8]

THE RESPIRATORY SYSTEM

Careful attention must be paid to the respiratory system in the neurosurgical patient. Mild respiratory insufficiency leading to modest hypoxia or hypercapnia can prove catastrophic. Hypercarbia increases cerebral blood flow and intracranial pressure; a 1 mm Hg rise in $Paco_2$ causes a 2.5% increase in cerebral blood flow[6] (Fig. 1). Residual narcotic or muscle relaxant effects are potential causes of respiratory depression. Small doses of naloxone (0.08 to 0.1 mg) and/or an anticholinesterase agent may be indicated.

Hypoxia may cause hyperemia and lactic acidosis, which will accentuate edema formation. It may occur secondary to pulmonary pathology such as aspiration, intraoperative air embolism, or pulmonary edema from neurogenic or other causes.[5] Postoperative administration of oxygen is prudent to prevent the development of diffusion hypoxia.

Surgical involvement in regions near the respiratory centers or compression of the brainstem from edema or hemorrhage can lead to respiratory abnormalities. Patients who are not awake and breathing adequately at the end of surgery should remain intubated. Occasionally patients with posterior fossa lesions may have lower cranial nerve dysfunction, causing vocal cord paralysis, swallowing difficulty, and partial airway obstruction.[20] Before extubation there are certain measurements that should be determined (Table 2). Close observation for development of obstruction or loss of gag reflexes must follow, and the facilities for immediate reintubation must be available.

Oxygen must be available during transfer of neurosurgical pa-

Table 2. *Factors to Be Determined Before Extubation of the Trachea of a Neurosurgical Patient*

Responsive to commands	
Respiratory pattern	Regular
Respiratory rate	10–35/min
Tidal volume	>3.5 mL/kg
Vital capacity	>15 mL/kg
$Paco_2$	30–45 mm Hg
Pao_2 (Fio_2 0.3–0.5)	>100 mm Hg

tients to the PACU. If the patient is to remain intubated, frequent evaluations of ventilation and oxygenation are necessary. Pulse oximetry is very useful and provides rapid data while arterial blood samples are being obtained.

Patients who require prolonged mechanical ventilation should not be allowed to "fight" the respirator or continue frequent coughing. Both situations can increase intracranial pressure. Patients may be given intravenous lidocaine (1 mg/kg) for cough suppression before gentle suctioning is attempted. If controlled or therapeutic hyperventilation is desired, patients may receive small amounts of sedation or muscle relaxants to facilitate this goal. Hyperventilation is most effective as an acute, short-term means of reducing intracranial pressure and is often combined with administration of steroids, diuretics, and possibly barbiturates.[3]

THERMOREGULATORY SYSTEM

Body heat loss can be extreme during extended surgical procedures. Resulting hypothermia may contribute to (1) shivering and rewarming, increasing oxygen consumption, (2) prolonged neuromuscular blockade, (3) vasoconstriction contributing to hypertension, and (4) delayed awakening from general anesthesia.[8] Hypothermia may be commonly associated with removal of a pituitary tumor, severe injury, or after spinal cord transection.[19]

Precautions should be taken intraoperatively to prevent heat loss via the use of humidified gases and warming blankets. Postoperative therapy consists of the use of radiant warmers, elevation of ambient temperature, warming of intravenous fluids, and humidification of inspired oxygen.

Hyperthermia is occasionally seen that is caused by autonomic disturbances such as injury to the floor of the fourth ventricle or thalamus and blood in the ventricular system. This may be accompanied by unresponsiveness, shallow respirations, hypotension, and tachycardia.[1] A surgical approach through infected tissue cavities such as sphenoid sinus or mastoid can increase the risk of postoperative meningitis. Malignant hyperthermia usually occurs intraoperatively but can reveal itself during the immediate postoperative period.

THE NERVOUS SYSTEM

Seizures occur in approximately 20% of neurosurgical patients postoperatively. A higher incidence arises in patients with brain abscess and is common after surgery near the cortical areas of the precentral and post-central gyri. In patients who suffered seizures preoperatively, this figure increases to 35%.[11] Seizures are also more likely to occur if the procedure involved a vascular lesion (intracranial aneurysm, arteriovenous malformation, hematoma).

The hazards of seizures include the risk of aspiration, increased incidence of neurologic deficits, possibly as a result of augmented cerebral edema, and increased oxygen consumption.[19] All patients who do not have adequate serum phenytoin levels preoperatively should receive doses of 7 to 15 mg/kg slowly, intravenously for prevention or treatment of postcraniotomy seizures. Careful monitoring of the electrocardiogram and blood pressure is necessary because arrhythmias or hypotension may ensue.[16]

Nerve palsies may be seen postoperatively due to the effects of malpositioning during surgery. Brachial plexus, sciatic, and peroneal nerve palsies have been described after craniotomy in the sitting position. Complete quadriplegia has also been reported due to extreme hyperflexion of the cervical cord in the presence of hypoperfusion or vascular occlusion.[10,21] Removal of cerebellopontine angle tumors may be associated with lower cranial nerve palsies (IX, X, XI, XII). Also carotid endarterectomy may produce dysfunction of cranial nerves VII, IX, X, and XII, causing interference with vocal cord function, gag reflexes, and inability to manage secretions. Cranial nerve and extremity function should be evaluated as soon as feasible after surgery.

DELAY IN REGAINING CONSCIOUSNESS

After any major intracranial surgery, the patient's return to consciousness is perhaps the most anxiously valued aspect of recovery. Altered states of wakefulness and motor or sensory effects seen preoperatively make it difficult to diagnose dysfunction noted postoperatively. Reactive swelling and consequent raised intracranial pressure are not infrequent accompaniments of neoplasms and abscesses.

If ischemia from increased pressure or reactive swelling occurs preoperatively or intraoperatively, recovery may be delayed or even limited. Patients who were intoxicated with alcohol or street drugs may show effects of drug interaction with anesthetics and thus a delay in awakening. Elderly patients may continue to experience the effects of preoperative medications (such as benzodiazepines) in the postoperative period.

If narcotics were used intraoperatively for maintenance of anesthesia, the patient may still be narcotized. "Pin-point" pupils and a decreased respiratory rate should indicate this. Small incremental doses of naloxone hydrochloride (0.05 to 0.1 mg) may be given intravenously until the desired response is obtained. Excessive doses must be avoided because of naloxone's ability to produce hypertension, tachycardia, and in some cases pulmonary edema secondary to catecholamine release.[2]

Hypothermia as well as hyperthermia may delay awakening and must be corrected. Derangements of water, electrolytes, and acid-base homeostasis are manifested frequently by altered sensorium, disorientation, or coma.

An intraoperative catastrophe such as a paradoxical air embolus may cause prolonged unconsciousness. This should be suspected after procedures performed in the sitting position.[10,21] Air is intrained into the venous system of a patient with a right-to-left shunt, via an atrial or ventricular septal defect, or a probe patent foramen ovale. As only small amounts of air in the arteries or brain stem can have devastating effects, the event might have passed unnoticed intraoperatively. Significant venous air embolism occurs in approximately 30% of patients in the sitting position; the complication may manifest as hypoxia postoperatively.[21]

Other intraoperative occurrences that delay recovery include cerebral infarction, intracranial hematoma, and cerebral edema. A small amount of air remains under the dura after any intracranial procedure. This volume depends on the extent of decrease in cerebral mass caused by hyperventilation, diuretic administration, and mass removal. After closure of the dura, the brain size increases as brain fluid reaccumulates, normocarbia returns, and the pressure within the air pocket increases. Tension pneumocephalus can cause a profound decrease in level of consciousness.[9] Diagnosis of this and other intracranial pathology is made with skull radiographs and computed tomography.

SPECIAL CONSIDERATIONS

Spinal Column Surgery

The level of the spinal column at which surgery has been performed determines the care necessary and the complications that may be encountered. Pain is often a significant problem in the early postoperative period, necessitating the use of narcotics for analgesia. The consequence of respiratory depression is not as severe as after craniotomy. Postoperatively the patient is nursed in a supine position, which improves venous drainage but decreases functional residual capacity. Pulse oximetry is indicated.[16]

Patients with traumatic spinal cord injuries are prone to many more complications. If there is complete transection around or just below C5, intercostal muscle activity is lost, and the patient is dependent on auxiliary means of increasing chest wall excursion. Occasionally, patients present with marginal preoperative respiratory embarassment. Careful assessment of ventilatory function is crucial before extubation is attempted.

Cardiovascular instability due to absent or decreased sympathetic tone leads to hypotension and bradycardia. Intravascular volume replacement often is used intraoperatively to treat the decrease in blood pressure. Because of reduced sympathetic effect, the vascular space is fully expanded and pulmonary edema is a common complication.[17] Direct measurement of pulmonary artery pressure via a Swan-Ganz catheter can detect early rises in pulmonary diastolic pressure before edema develops. Temperature regulatory mechanisms are impaired and hypothermia commonly results.

Aneurysm Clipping

One of the more common causes of deterioration after apparently successful aneurysm surgery is arterial vasospasm. Cerebral vasospasm as a complication of subarachnoid hemorrhage has been well documented. The pathogenesis of arterial vasospasm is still unresolved.[12] It appears to be an exaggerated reaction to vasoconstricting substances such as catecholamines, serotonin, angiotensin, calcium, bradykinin, and hemoglobin and its breakdown products.[16] If it existed preoperatively, it is more likely to prove problematic postoperatively.

Reversal of this decrease of cerebral blood flow by increasing systemic blood pressure and maintaining intravascular volume has proved useful. Nimodipine, a calcium channel blocker, has been found in clinical trials to decrease the incidence of cerebral vasospasm following subarachnoid hemorrhage, while causing few significant adverse effects.[13] It is likely that, in the future, many patients coming to surgery for aneurysm clipping following subarachnoid hemorrhage will be on Nimodopine therapy.

Posterior Cranial Fossa Surgery

Controversy concerning the use of the sitting position in neurosurgery continues. The advantages and disadvantages intraoperatively are currently debated in the literature.[10] Complications of the sitting position may include postural hypotension, arterial and venous air embolism producing hypoxia, cor pulmonale, and pulmonary edema. Cervical cord ischemia, midcervical quadriplegia, and lower extremity nerve palsies have also been reported.[1,21]

Advantages for the surgeon include an improved operating field with optimal venous drainage. For the anesthesiologist there is easy access to the airway and thorax. With appropriate monitoring and experience some neurosurgical centers report an excellent record of patient outcome after sitting position procedures.[10] Because of the manipulation of the brain stem in posterior cranial fossa surgery, prolonged ventilatory disturbances may occur.[20] In these situations, monitoring of ventilatory CO_2 response via capnography and other measures can help to evaluate the adequacy of ventilation before extubation. Lower cranial nerve dysfunction may cause vocal cord paralysis, swallowing difficulty, and airway obstruction. This may result in respiratory stridor, retained secretions, and the risk of aspiration. Pharyngeal and tracheal edema may develop during prolonged intubation in a flexed position, particularly if the prone or "park-bench" position is used. Prior to extubation, laryngoscopy will help to confirm the presence of adequate protective laryngeal function.

Hypophysectomy

The most important complication after hypophysectomy is diabetes insipidus. Patients develop massive polyuria (2 to 15 L/24 hours) during the operative procedure or, more commonly, in the postoperative period. This is accompanied by excessive thirst, dilute urine (<290 mOsm/kg), urine specific gravity <1.010, and serum sodium levels above 155 mg/dL. Therapy consists of administration of antidiuretic hormone as well as replacement of water and solute lost in urine. The use of aqueous vasopressin by the intramuscular or subcutaneous route in doses of 5 to 10 U leads to prompt antidiuresis for 4 to 6 hours.[8] For long-term management of central diabetes insipidus, the drug of choice is desmopressin (DDAVP), which is administered intranasally every 12 to 24 hours. Agonist therapy necessitates frequent monitoring of plasma electrolyte levels and osmolality with appropriate adjustments in fluid intake.

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Significance and Recovery Room Management of Postanesthesia Hypothermia and Shivering

*Richard B. Lilly, Jr., MD**

Studies have shown that about 60% of all patients arriving in the recovery room are significantly hypothermic.[24] A survey of patients' impressions of their recovery room stay showed that their greatest recollection was of "freezing to death." Conahan's work[3] documenting the advantages of heated humidification in some ambulatory surgery patients shows that the problem of hypothermia is not confined to major surgery, but is just as significant in the day surgery unit.

This review discusses the etiology and pathophysiology of postoperative hypothermia and concentrates on the significance and management of hypothermia in the recovery room.

ETIOLOGY

Mechanisms of Heat Loss

There are several mechanisms of heat loss from the anesthetized body. *Radiation* is the loss of electromagnetic energy from the body to colder objects in the room not touching the body. It is the largest source of heat loss, accounting for 65% of the body's losses. *Convection* is loss of heat to air currents and accounts for 25% of heat loss. High air flows and poorly located air conditioning vents that blow cold air on the patient add to this loss in the operating room. *Conduction* is loss of heat to surfaces the body is touching. The insulating value of mattresses should minimize this loss in the operating room. However,

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wetness increases conductive losses 25- to 30-fold and most patients are wet by the time the surgical preparation is completed.

Poikilothermia

Traditional teaching has been that anesthetized patients become poikilothermic. This means they are unable to actively regulate their body temperature, which consequently drifts passively toward ambient temperature. Vasodilatation early in the anesthetic brings heat from the core to the surface where it is lost to the environment through the mechanisms discussed previously.

More recently, Sessler[17, 18] has shown that active thermoregulation in the form of peripheral vasoconstriction occurs in patients anesthetized with halothane or fentanyl/N_2O. The temperature threshold at which active thermoregulation replaces poikilothermia is around 34.5°C. This compares to the awake patient in whom a 0.1° temperature decrease in the brain produces active thermoregulatory measures.

Nevertheless, 34.5° is significant hypothermia, and the vasoconstriction that does occur beginning at 34.5° may be deleterious to distal extremity perfusion and may interfere with pulse oximeter function. Furthermore, patients arriving in the recovery room with a temperature of 34.5° will suffer all of the adverse hemodynamic and metabolic consequences of rewarming discussed below. Therefore, despite Sessler's very interesting work, the prudent clinician will still treat patients as if they were poikilothermic and take all possible measures to keep them warm.

Cold Operating Rooms

Normal operating room temperature is usually around 20°C. Yet several studies have shown that 100% of anesthetized patients become hypothermic in rooms at 20°C or less.[14, 25] Room temperatures must be increased to 24° to 26° to prevent significant heat loss. An awake, naked human must expend energy to maintain body temperature when ambient temperature is less than 27° to 33°.[13] Therefore, patients will begin to cool or to shiver and vasoconstrict in the average operating room even before anesthesia is induced. Since the average recovery room temperature is probably only 22° to 23°, inadequately covered patients will have to expend energy to maintain their temperature even if they are not cold on arrival in the recovery room.

Greatest Heat Loss in the First Hour of Surgery

During the first hour of surgery we bring a patient into a cold operating room; take off most of his clothes and bedding; wet him with a preparatory solution; require him to warm and humidify a cold, dry breathing system; and anesthetize him, greatly reducing his ability to regulate his temperature. It is no wonder that his temperature falls rapidly. As surgery progresses the patient is draped, which greatly reduces radiant and convective losses. He warms the circle system, which reduces respiratory heat loss. And according to Sessler, he begins to regain thermoregulatory control as his temperature falls. Thus, the rate of heat loss decreases and may plateau as the case progresses. The clinical significance of this is twofold. First, it is clear that the management of recovery room hypothermia must begin as soon as the patient enters the operating room. Second, since most of the heat loss is early in the course of the procedure, it follows that patients having short, relatively minor surgery are at significant risk of hypothermia.

Spinal and Epidural Anesthesia

A comparison of similar groups of patients, one of which had spinal or epidural anesthesia and the other of which had general anesthesia, revealed that both groups arrived in the recovery room with the same degree of hypothermia. However, the general anesthesia group rewarmed twice as fast as the regional group.[24] The reasons seem obvious: the flaccid muscles and vasodilatation of the regional group persist into the recovery room and therefore heat loss continues until the block wears off. On the other hand, the patients in the general anesthesia group begin to actively rewarm themselves as soon as they emerge. Another study suggests that the rate of heat loss is even greater under epidural than general.[23] The clinical lesson for the recovery room care of patients is that one must pay careful attention to heat conservation measures in recovery room patients who have had major regional anesthesia.

Epidural narcotics have effects on shivering and heat regulation. Fentanyl and especially sufentanyl when administered epidurally appear to depress shivering and thermoregulatory ability in a dose-dependent fashion. Sevarino et al[19] have reported that 50 to 100 μg of epidural sufentanyl prevents shivering but also causes severe hypothermia to develop in the recovery room after cesarean section. Lower doses of epidural sufentanyl lacked this hypothermic effect. Matthews and Corser[12] have successfully used relatively much smaller doses of epidural fentanyl (25 μg) to treat the shivering that often develops in parturients during epidural anesthesia. Thus, further work needs to be done to clarify the relationship between epidural narcotics and hypothermia. They may have a role in treating shivering, but they may also be a cause of hypothermia in the recovery room.

PATHOPHYSIOLOGY

The adverse effects of hypothermia in the recovery room relate primarily to shivering, oxygen consumption, and the cardiovascular system. There are also significant effects on clotting, metabolism, and the central nervous system.

Cardiovascular System

Hypothermia causes peripheral vasoconstriction, which can increase blood pressure, cardiac afterload, and myocardial oxygen consumption. Fluid shifts from the vascular to the extracellular space. For every degree centigrade of hypothermia, 2.5% of the intervascular volume may be lost. The ensuing increase in hematocrit adds to the afterload increases. Cold-induced diuresis also adds to this relative hypovolemia. As the patient rewarms in the recovery room he will vasodilate and will require significant volume infusion to maintain filling pressures and cardiac output. Failure to give adequate intravenous fluid replacement during rewarming may cause significant hypotension.

With more severe hypothermia, progressive bradycardia, myocardial depression, and hypotension may occur. These problems are more common in severe exposure hypothermia than in moderate postanesthesia hypothermia.

The electrocardiogram (ECG) shows initial sinus bradycardia followed by prolongation of the PR and QT intervals with widening of the QRS complex. Ventricular irritability begins around 30° and ventricular fibrillation occurs between 25° to 28°. A cold heart is particularly sensitive to mechanical stimulation and cardiopulmonary resuscitation attempts could convert severe bradycardia to refractory ventricular fibrillation.

Shivering and Oxygenation

Postanesthetic shivering is a common complication. It can cause serious cardiorespiratory complications as well as patient discomfort, dental damage, increased intraocular pressure,[11] and disruption of delicate surgical repairs.

As little as a 0.3° drop in temperature increases O_2 consumption by 7%, a 1° drop increases consumption 92%, and the violent shaking seen in some emerging patients increases O_2 consumption by 500 to 700%.[10, 13]

All postanesthesia shivering is probably not caused by hypothermia. Sessler et al[16] observed that some normothermic patients shiver violently during emergence from anesthesia, and that some hypothermic patients do not shiver at all. They compared the electromyogram (EMG) results of patients shivering during emergence with those of nonanesthetized patients experiencing thermoregulatory shivering and with those of patients having pathologic clonus secondary to spinal cord injury. The postanesthesia shivering EMGs resembled those of pathologic clonus and were different from thermoregulatory shivering. This led to Sessler's conclusion that postanesthesia shivering is caused by "the spinal cord waking up before the brain does." This allows spinal reflexes that would normally be inhibited by the brain to become manifest. These exaggerated spinal reflexes need an initiating stimulus and Sessler postulates that "stimulation of cutaneous cold receptors could be one such trigger." If that is true, it has interesting therapeutic implications, as will be discussed in the section on therapy. Sessler et al[16] go on to describe three phases of emergence from isoflurane anesthesia: early recovery when brain and cord are equally anesthetized and there is no shivering; an intermediate stage (isoflurane concentration of 0.1 to 0.19% et) during which the exaggerated spinal reflexes cause shivering but thermoregulatory response is inhibited; and a final stage (isoflurane <0.1% et) when thermoregulation is no longer inhibited but spinal reflexes are now appropriately inhibited by the awake brain. Shivering in this late phase is all attributed to hypothermia.

Clinical Significance of Shivering

Whether postanesthesia shivering is caused by hypothermia, unopposed spinal reflexes, or a continuum of the two, the clinical significance is the same. Shivering causes dramatic increases in muscle metabolism and therefore increases oxygen consumption and the demand for cardiac output and ventilation. Respiratory acidosis will occur if, because of disease or residual anesthesia, the patient is unable to increase his ventilation. Likewise, if he is unable to increase his cardiac output, metabolic acidosis and venous oxygen desaturation will occur. Venous desaturation combined with ventilatory depression will cause arterial hypoxia. *Thus, severe postanesthesia shivering can be a direct cause of immediate postoperative hypoxemia and acidosis. Furthermore, the increased demand for cardiac output could push a patient with marginal myocardial or coronary reserve into congestive heart failure or myocardial ischemia.* Careful monitoring of blood gases and mixed venous O_2 saturation in rewarming patients is necessary to detect acidosis, hypoxia, and venous desaturation.

Fever is a resetting of the body's thermostat to a new, higher level. Actively cooling a febrile patient without first resetting his thermostat to more normal levels with an antipyretic drug will cause him to shiver even though his temperature is above normal. This can bring about all of the negative metabolic and cardiorespiratory consequences of shivering discussed previously. Before instituting active cooling routinely in a seriously ill, febrile patient with limited cardiorespiratory reserve, the pros and cons of active cooling should be considered.

Coagulopathy

Coagulopathy is a consequence of postoperative hypothermia that may be of more direct concern to the surgeons than the metabolic and cardiorespiratory problems already discussed. As such, it is a useful argument to use when pointing out to surgeons the need for various intraoperative warming measures they might otherwise find inconvenient.

All of the humoral clotting factors have decreased activity as the patient cools, just as all other metabolic processes slow down. The major cause of the coagulopathy, however, is sequestration of platelets in the liver. Although sequestration is the major effect, there is also some diminution in platelet function. When the patient is rewarmed, the platelets are released back into the circulation and within 2.5 hours their function returns to normal.[13]

Renal Effects of Hypothermia

Hypothermia induces a diuresis that contributes to the hypovolemia discussed previously. The kidney may interpret the vasoconstriction caused by hypovolemia as volume overload and respond with a diuresis. Also, glomerular filtration is reduced but tubular reabsorptive function is even more depressed, and this leads to a diuresis of dilute urine.

Neurologic and Metabolic Consequences of Hypothermia in the Recovery Room

Hypothermia slows all metabolic processes by a factor of about 7% per degree centigrade of temperature decrease. Liver blood flow is also reduced. The effects of all drugs dependent on liver metabolism for elimination are prolonged. The effects of nondepolarizing relaxants are increased. The minimun alveolar concentration (MAC) is decreased by 7% per degree centigrade. Consequently, if appropriate changes of inhalation anesthetic concentration are not made as a patient cools, he may be excessively deep on arrival in the recovery room.

At 33°C shivering stops and the patient can no longer actively rewarm himself. Semiconsciousness begins at 33° and coma develops by 30°. Short-term memory begins to decline at 35° and is decreased by 70% at 34°.

All of these facts taken together mean that significant postoperative hypothermia may cause serious delays in emergence from anesthesia.

RECOVERY ROOM TREATMENT OF HYPOTHERMIA

Prevention is always better than treatment. Aggressive measures taken in the operating room to prevent heat loss will pay off in the recovery room in terms of decreasing the adverse consequences of

Table 1. *Intraoperative Heat Conservation Measures*

1. Warm the operating room if possible to 23°–24° until the patient is fully draped
2. Keep as much of the patient covered as much of the time as possible. "Space Blanket" material may be helpful but lacking that, simple cotton operating room blankets are effective in preventing radiant heat loss. It is especially important to keep the patient's head and shoulders well covered
3. Use radiant heat lamps on exposed areas of the patient's skin during catheter placement, prepping, draping, and during the procedure when feasible
4. Use heated humidifiers. They can actually rewarm a cold patient. Heat and moisture exchangers may help prevent heat loss but are not helpful in actively heating a patient
5. Use intraveneous fluid and blood warmers
6. There is no evidence that water-circulating heating blankets placed under adult patients do any good. One study suggests they may have some synergistic benefit when used with heated humidifiers

hypothermia already discussed. Remember that the greatest heat loss is in the first hour in the operating room and if you wait until the patient begins to cool off it is already too late. Remember also that aggressive heat conservation may be just as beneficial to a 1-hour ambulatory surgery center case as it is to an abdominal aneurysm resection. In Table 1 I have listed, in order of importance, measures that are useful in the operating room to prevent and treat hypothermia. These are covered in detail in my earlier review of the subject.[9]

Supplemental Oxygen

The primary message of this review is the deleterious effect that hypothermia has on oxygenation in the recovery period. Therefore, the first treatment modality for a hypothermic patient is supplemental oxygen provided until the patient is fully rewarmed and maintaining normal O_2 saturation breathing room air. The O_2 should be started in the operating room before the trip to the recovery room if the patient is cold or shivering.

Severely hypothermic patients or moderately hypothermic patients who have compromised physical status should be kept asleep, intubated, and paralyzed until they are warmed fully. That will avert the adverse metabolic and hemodynamic consequences of rewarming and shivering.

Radiant Heat Source

Sharkey et al[20] have clearly demonstrated that a radiant heat source is very effective in stopping postanesthesia shivering. Radiant heat lamps directed at the face, chest, and upper abdomen stopped shivering within 60 seconds. Particularly significant is that the shivering stopped well before the core temperature rose. When the lights were turned off, shivering recurred rapidly but stopped again as soon as the lights were reapplied.

Others have used radiant heat after cardiac surgery and demonstrated decreased shivering, decreased O_2 consumption, CO_2 production, and ventilation requirement when compared with a control group who did not receive radiant heating.[8] In a similar study on young gynecology surgery patients, radiant heat in the recovery room decreased shivering, O_2 consumption, and catecholamine levels. Patient comfort was increased.[5]

The radiant heat source can be a simple apparatus of heat lamps on a stand or a more elaborate, commercially available radiant heat panel. The latter come as portable units or they may be ceiling mounted on tracks to be rolled over a patient's bed as needed. The heat source should shine on the patient's bare skin or at most through a thin sheet. One must be careful in uncovering a patient to use radiant heat that the patient is not then exposed to additional convection cooling because the recovery room is too cool and drafty. Burns may result if the radiant heat source is too close to the patient's skin.

I know of no studies that look at the value of radiant heat in postanesthesia shivering unrelated to hypothermia. However, based on Sessler's theory discussed previously that cutaneous cold receptors are the stimuli for exaggerated spinal reflexes during emergence, it would seem likely that radiant heat is beneficial in that situation as well.

Blankets

There are conflicting data as to the efficacy of simple cotton blankets in treating shivering. If warmed blankets are used, they may act as a radiant heat source and thus be useful if they are changed as soon as they cool to ambient temperature. On the other hand, room temperature blankets or a single warmed blanket that is not replaced frequently are not very effective in stopping shivering.

Circulating-water heating pads are of little value under the patient because of limited surface contact and low blood flow through the area of contact. If such a warming blanket is placed on top of a patient, it may serve as an effective radiant and conductive heat source.

Meperidine Stops Shivering

Numerous drugs have been used to treat postanesthesia shivering, including chlorpromazine, methylphenidate, diazepam, and various narcotics. None have been shown to be effective except meperedine. However, meperdeine is dramatically effective in very small doses. Claybon and Hirsch[2] reported "a high success rate and low incidence of adverse reactions using 12.5 to 25 mg of intravenous meperedine to stop postanesthesia shivering." If the patient had previously received naloxone, meperidine still successfully controlled the shivering, but 50 mg was required. There was no increased incidence of nausea or vomiting or respiratory depression. Subsequently, others have shown that meperedine also ameliorates the adverse hemodynamic and metabolic consequences of shivering.[15] Guffin et al[4] noted significant falls in mixed venous O_2 saturation in patients shivering after cardiac surgery. Treatment with 25 to 50 mg intravenous meperedine caused the venous saturation to return to close to preshivering levels. Macintyre et al,[10] using 25 mg, intravenous meperedine not only stopped shivering but also documented decreased O_2 consumption and CO_2 production after the shivering was controlled in a group of noncardiac surgery patients. Meperedine has also been used successfully to treat shivering during epidural analgesia for cesarean section.[1]

In all studies of the effect of meperedine on shivering, the onset of action was within 5 minutes, often faster. The duration of effect was long enough to allow the patient to rewarm; therefore recurrent shivering is not likely. None of the studies looked specifically at the differences in meperedine's effect on hypothermic compared with normothermic postanesthesia shivering.

No studies have compared other methods of treating postanesthesia shivering, like radiant heat with meperedine. However, in my opinion, small doses of intravenous meperedine are the most effective and rapid way to control shivering and its adverse effects on oxygenation.

Special Considerations after Cardiac Surgery

After hypothermic cardiopulmonary bypass, patients tend to arrive in the recovery room hypothermic. Their core temperature is often not fully restored even though the blood temperature may be 37°C when weaning from bypass. Therefore, their temperature drifts down during wound closure and the early recovery period.

If they are not fully paralyzed, they may shiver in the recovery room. Sladen et al[21, 22] reported two phenomena. If the patients shivered in the recovery room, the shivering interfered with the mechanical ventilator and the patients tended to be underventilated. On the other hand, paralyzed hypothermic patients were overventilated when cold and underventilated as they rewarmed, unless there was careful adjustment of ventilation during rewarming in the recovery room. The rewarming period for recovering open heart surgery patients was 12 hours and there was an overshoot to above normal temperatures at the end of the period.

Increased O_2 consumption, energy expenditure, CO_2 production, and venous desaturation have all been reported during postoperative rewarming of cardiac surgery patients.[4, 7] Holtzclau and Green[6] noticed that masseter muscle fasciculations precede these deleterious respiratory and metabolic phenomena.

There are a couple of clinical messages in these studies of cardiac surgery patients. First, they are clearly at increased risk of postoperative hypothermia and its attendant cardiorespiratory consequences.

It is prudent to give these patients an extra dose of relaxant prior to transport to the recovery room and to keep them paralyzed until they are fully warm. Radiant heat sources, intravenous meperedine, and prolonging bypass to allow full core and rewarming have all been effective in decreasing postoperative increases in energy expenditure due to rewarming. Second, even though the work was done on cardiac surgery patients, the lesson applies to any sick, intubated, mechanically ventilated patient who is cold after surgery.

Other Recovery Room Measures

If large volumes of blood and fluid replacement are needed in the recovery room, the use of fluid warmers is just as important as in the operating room. There are now highly efficient fluid warmers available that allow the fluid path tubing to be disconnected from the machine in the operating room and reconnected to a new warmer in the recovery room. Adoption of this type of system will facilitate continued use of the fluid warmer in the recovery room.

Ambient temperature is important. As noted prevoiusly, patients who have residual spinal or epidural anesthesia or who have had some epidural narcotics may remain somewhat poikilothermic in the recovery room. Many patients are poorly covered in the recovery room. Most patients have a subjective feeling of being cold during emergence. For all these reasons a comfortably warm room temperature is important.

SUMMARY

Hypothermia is very common after anesthesia and surgery. Rewarming increases O_2 consumption often dramatically. Shivering after anesthesia is partially related to hypothermia and partially to spinal reflexes unopposed by a still anesthetized brain. Whatever the etiology of the shivering, it is very costly in terms of O_2 consumption and cardiac output.

Hypothermia should be prevented in the operating room. In the recovery room, hypothermia and shivering must be treated aggressively to avoid the adverse hemodynamic and metabolic consequences. The two most effective treatment modalities are intravenous meperedine and radiant heat. Supplemental O_2 is required until shivering stops and body temperature is normal. Seriously hypothermic patients should remain intubated and paralyzed until they are warm to decrease the metabolic and cardiorespiratory costs of rewarming.

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Diagnosis and Treatment of Hyperthermia in the Postanesthesia Care Unit

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Fever has been regarded as a cardinal manifestation of disease since ancient times. From its first description by Hippocrates as an ill humor, yellow bile associated with fire, fever has stirred the imagination of poets, scholars, and scientists.[1, 24]

Fahrenheit developed the first effective thermometer in the 18th century. The first scientific measurement occurred in 1776 when John Hunter invented the mercury in glass thermometer, which was placed under the tongue. Liebig and Lavoisier determined that fever represented the heat generated by metabolism. Bernard discovered heat loss to be regulated by the nervous system, which produced homeostasis of body temperature.[1]

After making 25,000 measurements and observing temperature variations in 32 disorders, Wunderlich in 1868 postulated that an abnormal temperature was a sign of disease.[24] In 1875 von Liebemeister observed that body temperature was regulated at a higher level during fever, forming the basis for the theory of the hypothalamic set point.[1]

PATHOPHYSIOLOGY

Although often used interchangeably, a distinction should be made between "fever" and "hyperthermia."[37, 48] Fever is an endogenously triggered process with metabolic and functional changes resulting in an alteration in the hypothalamic set point.[14] Treatment is

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with drugs, such as antipyretics. Hyperthermia is an exogenously triggered process in which body temperature rises, but the set point does not change. It represents a failure of thermoregulation. In hyperthermia the increase in body temperature overrides the ability to lose heat.[14] Treatment is primarily by cooling and promoting mechanisms for increased dissipation of heat.

Body temperature is usually maintained within a narrow range of 37 plus or minus 1°C via feedback mechanisms.[5, 37] Heat production normally equals heat dissipation.[48] The upper lethal limit of body temperature is 42 to 43°, the "thermal maximum." At greater than 40°, delirium, convulsions, and rigors develop. At greater than 42°, as a result of heat injury itself, irreversible changes of coma, cerebral hypoxia, acidosis, rhabdomyolysis, dehydration, denaturation of proteins, and death ensue.[15] Infants, the elderly, and debilitated persons are most subject to body temperature aberrations.[15] In some pathologic conditions central temperature control is lost, and extreme temperatures may develop.

Fever is usually associated with microbial agents or other disease processes stimulated by drugs, allergies, or immune conditions which trigger the release of a substance that acts directly on the thermoregulatory center located in the anterior hypothalamus and results in an increase in set point for body temperature.[5, 14, 15, 48] In the case of microbial agents, endogenous pyrogen (interleukin 1) is released from monocytes and macrophages[1] and works through monoamines, prostaglandins, and cyclic nucleotides to activate host defense mechanisms and maintain body temperature at an increased level.[5, 14, 15, 48]

The significance and purpose of fever is controversial. There is uncertainty as to whether it should be ablated or encouraged.[14, 15] It has been reported to confer survivial benefit by increasing resistance to infection via activation of lysosomal enzymes.[5] Ability to mount a fever within 24 hours in the face of infection is said to improve prognosis.[15, 18]

MECHANISMS

Fever results from heat production in excess of heat loss (Table 1). Mechanisms of heat gain include (1) basal metabolic rate, to which the main contributions are the metabolic activity of the heart and liver at rest; (2) skeletal muscle contraction and shivering ("chills"), which may increase heat production 10 to 20 times and is the primary means of heat production; (3) hormonal secretion of thyroxine and epinephrine; (4) nonshivering thermogenesis via brown fat, occurring mainly in infants; and (5) exogenous heating from a warm environment.[19, 48]

Mechanisms of heat loss include (1) radiation from skin, account-

Table 1. *Regulation of Body Temperature*

- Internal mechanisms
  - Hypothalamic temperature regulating center*
  - Autonomic nervous system*
  - Basal metabolic rate, liver and heart
  - Skeletal muscle contraction and shivering
  - Hormonal secretion, thyroid and adrenal
  - Brown fat
  - Sweat glands
  - Blood vessels, vasomotor activity
- External mechanisms
  - Radiation
  - Conduction
  - Convection
  - Evaporation

* The hypothalamic temperature regulating center and the autonomic nervous system regulate body temperature internally. Additional mechanisms allow for exchange between the body and the environment.

ing for 70% of heat loss; (2) conduction; (3) convection; and (4) evaporation from skin and lungs, influenced by humidity, accounting for about 30% of heat loss.[19, 48]

Central and peripheral receptors interact with the autonomic nervous system to regulate body temperature. Short-term, neural influences predominate, while long-term, hormonal influences prevail. An increase in core temperature activates the autonomic nervous system to promote vasodilatation and sweating.[19]

EFFECTS ON ORGAN SYSTEMS

Physiologic effects of hyperthermia place stress on the cardiovascular and respiratory systems of the individual. Oxygen consumption increases 7% for each degree Fahrenheit increase in temperature.[15] The major cardiovascular effects are vasodilatation, increase in pulse rate, decrease in venous return, and hypotension.[15] Arrhythmias, congestive heart failure, and ischemia may result, especially in someone with an already compromised cardiovascular system.[19]

The major respiratory effects are decrease in tidal volume and increase in frequency of respiration, increase in ventilatory response to hypercapnia and hypoxia, increase in hypoxic pulmonary vasoconstriction, and shift of the oxyhemoglobin dissociation curve to the right.[19]

Endocrine effects include mobilization of free fatty acids and lipolysis. There is glycogen depletion and excess lactate production.[19] Fluid and electrolyte abnormalities include increased sweating resulting in dehydration; alterations in sodium, potassium, and chloride balances; hemoconcentration; and azotemia.[19] Central nervous system effects may include seizures, delirium, and coma.

DIAGNOSIS AND TREATMENT

Evaluation of fever requires documentation, assessment of clinical significance, and discovery of the underlying cause. Careful monitoring and general cardiovascular, respiratory, and metabolic support are indicated.

History, physical examination, roentgenograms, cultures, complete blood count, electrolytes, renal and liver function tests, coagulation studies, muscle enzymes, and arterial blood gases should be obtained. Vital signs, urine output, and neurologic status should be observed.[48] The general treatment for hyperthermia is promotion of vasodilation, sweating, and evaporation.[5] Fluids, cooling measures, and antipyretics may be used. Cooling measures such as sponging with tepid water or alcohol and application of blankets, fans, and ice packs are useful.[48] Salicylates, acetaminophen, nonsteroidal anti-inflammatory agents, and steroids reduce fever by inhibition of prostaglandin synthesis or release.[48]

The outcome will depend on the etiology, general condition of the patient, and the level of temperature reached. Overzealous treatment may produce hypothermia, which is also undesirable. The temperature should be reduced to within 2° of normal to maintain baseline tissue oxygen demand and carbon dioxide production.

SPECIFIC CONDITIONS PRODUCING HYPERTHERMIA

A number of pre-existing medical diseases or events related to anesthesia and surgery may result in hyperthermia in the postanesthesia care unit (PACU) (Table 2). The anesthesiologist is often presented with a diagnostic dilemma, since the manifestations of several of these conditions such as malignant hyperthermia, thyroid storm, pheochromocytoma, and sepsis may be similar. Endocrine abnormalities, the presence of infection, recreational drug use, drugs or blood administered in the operating room, climatic conditions within the operating room, or nervous system dysfunction may all produce postoperative temperature elevations. The anesthesiologist must distinguish among these.

Causes may be as simple as a pre-existing upper respiratory tract infection or the result of extensive surgery with associated tissue

Table 2. *Conditions Causing Hyperthermia in the PACU*

Malignant hyperthermia
Thyroid storm
Pheochromocytoma
Sepsis
Drug reactions
Reactions to blood and blood products
Central nervous system catastrophe
Vomiting and aspiration
Exogenous hyperthermia

trauma, or as extreme as a catastrophic central nervous system hemorrhage or sepsis. Forty per cent of postoperative patients develop fever. Resolution of the fever requires appropriate medical management based on the diagnosis.

Malignant Hyperthermia

Incidence. Heading the list of conditions likely to produce hyperthermia in the early postoperative period is malignant hyperthermia. Although this syndrome is more often manifested in the operating room, it may occur in the recovery room and even later in the postoperative course.

Malignant hyperthermia is defined as a pharmacogenetic disorder resulting in an abnormal reaction to anesthetic drugs due to a genetically determined metabolic defect involving muscle. The key elements are its relationship to anesthetic drugs, heredity, metabolism, and muscle.[17, 41, 42]

The incidence of malignant hyperthermia during or following anesthesia is 1:15,000 in children and 1:20,000 to 1:50,000 in adults. The incidence is variable depending on specific patient populations. Malignant hyperthermia affects all races, both sexes, and primarily the young.[17, 41, 42] Although the mortality has been significantly reduced since the availability of dantrolene in the late 1970s, deaths still occur. The genetic pattern, which has not been fully elucidated, is autosomal dominant, with reduced penetrance and variable expressivity.[17, 41, 42]

Diagnosis. An episode of malignant hyperthermia is characterized by a rapid and progressive rise in body temperature and biochemical changes indicative of a hypermetabolic state. It may be accompanied by jaw or total body muscular rigidity. The syndrome is usually triggered by potent anesthetics and muscle relaxants, most frequently halothane and succinylcholine. Other drugs such as catecholamines, belladonna, anticholinesterases, calcium, potassium, digitalis, theophylline, and ketamine are thought to play a role either in triggering or sustaining an episode. The stress of surgery, anesthesia, and preceding trauma may contribute.[17, 41, 42]

Although the characteristic clinical triad consists of fever, rigidity, and metabolic acidosis, many cases are equivocal. At the cellular level, there is loss of regulation of intracellular ionized calcium. Increased anaerobic and aerobic metabolism and hydrolysis of high-energy phosphate bonds produce the very high temperature. A drug such as dantrolene, which lowers intracellular calcium by preventing its release from the sarcoplasmic reticulum, improves the situation.[17]

The clinical signs of malignant hyperthermia are tachycardia, tachypnea, arrhythmias, rigidity, cyanosis and mottling, blood pressure lability, profuse sweating, and rapidly rising and sustained fever.[6, 17, 22, 41] The tachycardia may often be "unexplained." Arrhythmias, supraventricular or ventricular, are secondary to the biochemical derangement. These signs may have a more subtle appearance in the recovery room than in the operating room and therefore are often missed. Manifestations may also reappear in the PACU following successful treatment of an acute episode in the operating room. Lack of vigilance and a false sense of security in the PACU have led to treatment which was "too little, too late," with serious morbidity and mortality.

The laboratory findings of malignant hyperthermia are hypercarbia, hypoxemia, metabolic and respiratory acidosis, hyperkalemia and other electrolyte abnormalities, myoglobinemia/myoglobinuria, and elevated creatine phosphokinase (CPK) level.[6, 17, 22, 41] An arterial blood gas analysis is very useful in supporting the diagnosis. Serum electrolyte and enzyme counts should be obtained. Myoglobinura and elevated CPK are later manifestations of muscle necrosis. The CPK may not peak until 8 to 12 hours post-episode.[6, 17, 22]

Monitoring should be established to include electrocardiography, blood pressure, temperature, urinary output, arterial line, central venous pressure, and pulse oximetry. If the patient is on a mechanical ventilator or if otherwise feasible, capnometry should be employed.[6, 7]

Treatment. The treatment includes the following.[7,17,41,42]

Dantrolene (1 to 10 mg/kg intravenously), which is the specific antidote for malignant hyperthermia, should be administered immediately. It is supplied in the form of a lyophilized, orange powder that is relatively insoluble. Each 20-mg ampule also contains mannitol and sodium hydroxide to enhance solubility, reaching a pH of 9.5 when reconstituted.

The average effective dose to treat a crisis is 2.5 mg/kg intravenously. Distilled water, 60 mL, is added to the ampule of dantrolene. Warming, shaking, and the use of a dispensing pin facilitate rapid mixing. The drug may be administered at the rate of 1 mg/kg/minute beginning at a level of 2.5 mg/kg/minute until the parameters improve. The dose may be repeated in 15 minutes if necessary.

Oxygen. Since there is increased metabolic demand, supplemental oxygen should be provided.

Cooling Measures. These may consist of immersion in an ice bath or the use of cooling blankets. Ice packs may be placed over great vessels, and lavage of body cavities such as nasogastric or rectal lavage may be instituted. Iced fluids may be given intravenously. Care must be taken not to overcool.

Sodium Bicarbonate. This should be given to treat the metabolic acidosis, based on blood gas analysis.

Antiarrhythmics. Most arrhythmias respond to the treatment of acidosis. Persistent arrhythmias should be treated with procainamide or other antiarrhythmic agents. Lidocaine is now considered safe for the treatment of ventricular arrhythmias during a malignant hyperthermic crisis. Beta blockers may also be used. Calcium channel blockers should be avoided because they have been associated with electrolyte abnormalities and myocardial depression.

Maintenance of Urine Output. Urine output should be maintained at a level of 2 mL/kg/hour by the use of adequate fluids, furosemide, and mannitol.

Management of Hyperkalemia. Glucose, 50%, and insulin may assist in the treatment of hyperkalemia by shifting the potassium intracellularly.

The patient should be observed closely in a recovery room or intensive care environment until the danger of subsequent episodes is past, for at least 24 hours. Administration of additional dantrolene is recommended by intermittent bolus or infusion over 24 hours. Side effects of dantrolene include muscle weakness, difficulty with ambulation, vertigo, blurred vision, sedation, nausea, and vomiting. Phlebitis may develop if dantrolene extravasates from the vein.[6, 7]

Outcome. Complications of malignant hyperthermia include consumption coagulopathy, renal failure, pulmonary edema, muscle necrosis, and neurologic sequelae. Inadvertent hypothermia may develop from overzealous cooling.[6, 7]

Successful outcome depends on early recognition and prompt treatment. Mortality, once 70%, has been reduced to about 10%. Protocol, supplies, and especially dantrolene should be available in all anesthetizing and postanesthetic recovery locations. Close observation and monitoring should extend into the recovery room because malignant hyperthermia can first manifest after surgery and anesthesia and recrudescence of an acute episode following treatment may also occur.[6, 7] If the diagnosis is in doubt, it is safer to treat than to delay. The undesirable effects of dantrolene are minimal compared to the risk of serious morbidity or mortality.

Response to dantrolene does not confirm the diagnosis of malignant hyperthermia.[41, 42] Dantrolene has a nonspecific effect on lowering body temperature and may be used for emergency treatment of other catastrophic hyperthermic conditions. The patient who has experienced a malignant hyperthermic episode should subsequently be referred for muscle biopsy and contracture testing.

Thyroid Storm

Incidence. Another crisis that may appear in the recovery room is thyroid storm. Many of its features are similar to those of malignant hyperthermia.[39, 51] The incidence of storm is from 2 to 8% in patients with hyperthyroidism, affecting primarily females, in the third and fourth decades.[8, 50] The signs and symptoms, an exaggeration of those of thyrotoxicosis, are associated with an increase in circulating thyroid hormone and represent a decompensated state of hyperthyroidism.[3, 8, 50]

Elevated temperature may be present along with cardiovascular, gastrointestinal, and central nervous system changes.[3, 46, 52] Thyroid storm occurs rarely now as a result of improved treatment modalities for hyperthyroidism.[50] The preoperative administration of propranolol and other drugs, however, does not preclude the development of thyroid storm,[8, 53] which is likely to have an abrupt onset 6 to 18 hours postoperatively and will result in a 70% mortality untreated.[8, 50]

The crisis may occur because of inadequate preparation of a patient known to be hyperthyroid for surgery or an unrecognized diagnosis of thyrotoxicosis.[50] Apprehension, anxiety, iodine withdrawal, surgical stress, trauma, infection, or diabetes may precipitate or aggravate the problem.[8, 26, 35, 44] Sometimes it may be difficult to distinguish the signs and symptoms of hyperthyroidism from hormonal changes reflecting the body's reaction to illness or injury.

Diagnosis. Clinical signs and symptoms may include fever, warm and flushed skin, sweating, tachycardia, rapid atrial arrhythmias, hypotension, pulmonary edema, diarrhea, nausea and vomiting, jaundice, hyperglycemia, agitation, convulsions, or coma.[3, 20, 35, 38]

The temperature may reach 38 to 41°C. Hyperdynamic circulatory changes compensate for an increase in tissue oxygen demands.[36] The cardiovascular effects may be direct or mediated by catecholamines. Hyperglycemia is a result of impaired insulin secretion.[36] Excessive thermogenesis, hypermetabolism, and defective thermoregulation are contributory.[26, 36] Mental status is an important prognostic sign.[20, 50]

Diagnosis is made on the basis of clinical presentation. Thyroid function tests, total serum T4, T3, and thyroid-stimulating hormone, radioactive iodine, and thyroid scan are confirmatory.[8, 51, 52] Monitoring of electrocardiogram, blood pressure, temperature, and fluid balance must be undertaken. The placement of a Foley catheter, arterial line, and central venous pressure line or pulmonary artery catheter is desirable.[8, 26, 36, 52] The goals of treatment are to prevent further synthesis or release of thyroid hormone and to block its peripheral manifestations.[8, 26, 35]

Treatment. Treatment consists of the following.[8, 20, 26, 35, 36, 52]

Propylthiouracil. Propylthiouracil is given in a 900- to 1200-mg loading dose, then 300 to 400 mg every 8 hours orally or via nasogastric tube; or *methimazole*, 90- to 120-mg loading dose, is given, then 30 to 40 mg every 8 hours. These drugs prevent the synthesis of T3 and T4 by blocking the uptake and organification of iodine from the blood. They are effective within an hour of administration. Propylthiouracil also inhibits the conversion of T4 to T3, reducing its level by 50% in a day.

Sodium Iodide. Sodium iodide, 2 g intravenously saturated solution of potassium iodide, SSKI, or Lugol's solution, 30 drops orally may be administered. These medications inhibit the release of preformed thyroxine. Propylthiouracil should be given prior to their administration.

Propranolol. Propranolol, 1 to 2 mg intravenously every 5 minutes until symptoms are controlled or 120 mg orally acutely is administered. Subsequently, 40 to 80 mg orally every 6 hours may be given. This β-blocker counteracts the peripheral effects of thyroxine, reducing the heart rate, treating tachyarrhythmias, and improving hyperdynamic rate-related congestive heart failure. Propranolol also blocks conversion of T4 to T3. It should be given judiciously in patients with heart block, asthma, or congestive heart failure due to other causes.

Hydrocortisone. Hydrocortisone, 200 to 300 mg intravenously, is used to treat the relative adrenal insufficiency associated with thyroid storm. It also acts to inhibit hormone release, as well as conversion of T4 to T3, and exerts a cytoprotective effect.

Fluids, Electrolytes, Glucose, and B Vitamins. Dehydration must be corrected and excess energy and nutritional requirements met. Patients may require 4 to 5 L of crystalloid per day.

Cooling Measures. Hyperthermia may be treated with sponging, cooling blankets, and lavage.

Sedation. The patient should be maintained in a quiet atmosphere. Narcotics, barbiturates, benzodiazepines, and phenothiazines may be used for sedation. Chlorpromazine, 25 to 50 mg intravenously, is ideal to sedate and to prevent shivering induced by cooling measures. Sympathoadrenal stimulation should be avoided.

Digitalis, Diuretics. These may be used to treat congestive heart failure or pulmonary edema, which when induced by thyrotoxicosis, may be refractory to digitalis.

Oxygen. The patient should receive supplemental oxygen while experiencing a period of increased oxygen demand.

Acetaminophen. This drug may be given to control fever. Salicylates should be avoided since they bind with protein, thereby increasing free thyroxine levels.

Plasmapheresis or Dialysis. These modalities may be used for removal of the excess hormone in the absence of response to the above measures.

Successful management of thyroid storm, like malignant hyperthermia, requires close observation and intensive treatment. With improved methods of monitoring and pharmacologic therapy, a favorable outcome is likely.

Pheochromocytoma

Incidence. Pheochromocytoma is a catecholamine-secreting tumor arising from chromaffin cells of the sympatho-adrenal system.[32, 43] About 90% of tumors arise in the adrenal medulla, the others along the sympathetic chain in various locations.[43] The incidence of pheochromocytoma in the hypertensive population is from 0.1 to 0.5%,[34] and usually occurs in patients aged 30 to 50 years. A small portion, (10%), are inherited as part of a syndrome of multiple endocrine adenomatosis and are associated with other endocrine tumors.[43]

Pheochromocytoma is a great mimic,[4, 13] resembling malignant hyperthermia, renal or biliary colic, shock, or sepsis.[32] A crisis can be precipitated in the perioperative period by direct mechanical stimulation or manipulation of the tumor. Indirect release of catecholamines from the tumor may also result in massive hypertension and other effects.[52] Drugs that can bring this about include opiates, metoclopramide, adrenocorticotropic hormone, droperidol, iodine contrast, or any drug with the potential for causing histamine release.[2]

Diagnosis. Catecholamine excess as a result of tumor secretion increases the metabolic rate and thereby produces pyrexia, which is a frequent but not classic manifestation.[13] The traditional pheochromocytoma triad consists of headache, tachycardia, and diaphoresis.[52] Intense peripheral vasoconstriction, ischemia, and catastrophic cardiovascular collapse may ensue.[34]

Paroxysmal hypertension may alternate with hypotension.[34] The hypertension is caused by the release of norepinephrine and its effect on peripheral blood vessels.[33, 34] The hypotension may reflect a predominant effect of epinephrine on peripheral vessels or the relative hypovolemic state produced by chronic intravascular constriction with depletion of intravascular volume.[4] Postural hypotension, even syncope may develop.

Other signs and symptoms may include palpitations, nausea and vomiting, anxiety, dyspnea, abdominal or chest pain, or blurred vision.[38, 43] Hyperglycemia, acidosis, pulmonary edema, ventricular arrhythmias, cyanosis, and encephalopathy are characteristic.[33] Erythrocytosis, leukocytosis, myoglobinemia/myoglobinuria, and disseminated intravascular coagulation may be present.[27, 45] Renal failure may result from hypoperfusion due to extreme vasoconstriction or rhabdomyolysis.[33] Increased CPK is secondary to the ischemic changes occurring in skeletal and cardiac muscle.[45] Cardiomyopathy with congestive heart failure or myocardial infarction may develop.[45]

Ischemia induces cellular injury, releasing vasoactive substances and lactate.[27] Pressor doses of catecholamines produce ischemia of skin and muscle. Ischemia of the hepatic and splanchnic circulation affects the production and utilization of lactate.[33, 34] Excess lactate is also produced by glycogenolysis in the liver.[27]

In addition to the signs and symptoms, laboratory tests to confirm the diagnosis are the urinary metanephrine level, which is the most sensitive test, urinary normetanephrine and vanillymandelic acid, and plasma epinephrine and norepinephrine.[38, 43] Radiologic testing such as abdominal ultrasound, computed tomographic scan, and adrenal angiography may be adjuncts to diagnosis.[38, 43]

In patients with unsuspected pheochromocytoma, 50% of deaths occur at the time of anesthesia and surgical stress, or during pregnancy.[52] Invasive hemodynamic monitoring should be instituted. Any delay in diagnosis can result in rapid deterioration.

Treatment. The treatment regimen is the following.[27, 33, 34, 38, 43]

Adrenergic Receptor Antagonists. The alpha blockers phentolamine, given as a 0.5 to 1 mg bolus intravenously or by infusion at 1 to 10 μg/kg/minute, or phenoxybenzamine, 1 mg/kg intravenously over an hour, may be used acutely to produce alpha-receptor blockade. Phenoxybenzamine may continue to be administered in a dose of 20 to 80 mg/day. Reflex tachycardia may result.

Prazocin is a selective alpha-1 blocker that may be given in a dose of 0.5 to 1 mg intravenously or in doses of up to 20 mg orally.

The beta blocker propranolol, 0.5 to 1 mg intravenously, is used for acute treatment to a total of 0.1 mg/kg. This drug is given to control tachycardia and arrhythmias. It should only be used after the administration of alpha blockers, to prevent a marked increase in blood pressure due to beta-induced peripheral vascular constriction. Complications from beta blockade are heart failure and pulmonary edema. The drug may be continued chronically at a dose of 20 mg every 8 hours.

Esmolol, 50 to 500 μg/kg intravenous bolus up to 300 to 500 μg/kg/minute infusion, is a cardioselective beta blocker with a beta-1 predominant effect. Its rapid metabolism and 20-minute half-life allow for rapid termination of action.

Labetalol, is an alpha and beta blocker that may be given intravenously at a dosage of 2 mg/minute to a maximum dose of 300 mg. It must be given cautiously because of its beta effects. Reflex tachycardia does not occur.

Direct Vasodilators. Sodium nitroprusside, 20 to 50 μg bolus or intravenous infusion of 1 to 10 μg/kg/minute, is given acutely in combination with phentolamine to lower blood pressure. A direct arteriolar dilator with rapid onset, consistent effect, and short half-life, it maintains flow to vital organs. Coronary steal and cyanide toxicity with metabolic acidosis are side effects.

Nitroglycerine, 20 to 50 μg bolus or infusion of 1 to 10 μg/kg/minute intravenously, also lowers preload. It is less potent than nitroprusside. It dilates capacitance vessels, has a short half-life, and no toxicity.

Diazoxide, 300 to 600 mg intravenously, is a potent vasodilator, with a 5-minute onset and duration of several hours. Side effects include hypotension, tachycardia, ischemia, arrhythmias, hyperglycemia, and sodium and water retention.

Lidocaine, 50 to 200 mg intravenously, may be given to control ventricular arrhythmias.

Sodium bicarbonate is given to correct acidosis, thereby improving peripheral perfusion and oxygenation.

Fluids, whether crystalloid, colloid, or blood, are administered in large quantities to restore intravascular volume in the face of massive hypovolemia.

Cooling measures should be undertaken to treat pyrexia.

Steroids may provide membrane protection.

If the patient cannot be satisfactorily stabilized, emergency surgery to remove the tumor may be necessary. This situation is associated with increased risk of complications.[33, 34] Most patients respond to alpha blockers and vasodilators plus supportive measures. With the use of alpha blockers mortality has decreased from 60 to 6%.[52]

Sepsis

Incidence. The term *sepsis* encompasses the physiologic alterations and clinical consequences of micro-organisms or their byproducts in the blood stream or tissues.[18] In 20% of patients these events progress to septic shock, manifested by peripheral circulatory failure and cell death secondary to infection.[18]

Sepsis may be precipitated by trauma or surgery. Mortality, reported as 40 to 80%, will depend on the underlying disease, age of patient, and cardiovascular stability.[9] A third of infections are caused by gram-positive flora such as *Staphylococcus aureus* and *Streptococcus pneumoniae* and two thirds by gram-negative organisms.[9] There is an increasing incidence of infections due to resistant organisms such as *Klebsiella* and *Pseudomonas*.[9]

Patients who have undergone gastrointestinal or urologic surgery are susceptible, as well as those patients who have uterine infections or a perforated viscus.[9] Patients with underlying neoplasms, the immunosuppressed, and those who have indwelling catheters or who have been on mechanical ventilation are also likely to develop sepsis.[9, 23] Diseases such as cirrhosis and diabetes increase susceptibility.[9]

Diagnosis. Manifestations are often nonspecific and clinical clues subtle.[18] Particularly in the elderly, classical signs and symptoms may be absent.[23] Patients in the recovery room following surgical intervention for drainage of an abscess or removal of infected tissue may be on the brink of developing systemic manifestations of sepsis.[23]

Fever may be the initial predominant symptom. An increased temperature is the body's acute-phase response to infection and is mediated by exogenous or endogenous pyrogen.[15, 18] Along with fever there may be tachycardia and respiratory changes such as hyperventilation and respiratory alkalosis.[9, 18] These are stimulated by an increase in circulating catecholamines. Chills may accompany the fever when blood is shunted from the skin. The body's compensatory response is to increase heat generation from muscular activity. Early mental changes of confusion or agitation may progress to full-blown encephalopathy with seizures and coma.[52]

Hypotension plus vasodilatation are significant features of septic shock.[52] Vasodilatation is due to the release of vasoactive substances. Cardiovascular hemodynamics are characterized by a decrease in peripheral vascular resistance and an increase in cardiac output. The patient may appear flushed, with warm skin and increased pulse pressure.[52] As the pathophysiology progresses, there will be derangements in serum electrolytes, metabolic acidosis with increase in lactate levels,[52] ventilatory insufficiency with arterial desaturation and progression to acute respiratory distress syndrome, and renal insufficiency.[9, 18]

Blood pressure falls secondary to hypovolemia and myocardial dysfunction. Hypovolemia is the result of exudation of intravascular fluid, venodilation, and volume deficits. There is a decrease in mixed venous O_2 and arteriovenous O_2 difference.[18, 52] Coagulation abnormalities such as disseminated intravascular coagulation and thrombocytopenia may occur.[18] There may be leukocytosis with a shift to the left. Gastrointestinal bleeding and hyperbilirubinemia represent end-stage damage to other organ systems.[18] The breakdown of protein and hormonal changes result in muscle catabolism and myalgias.[18, 52] Death occurs as a result of refractory vasodilatation, severe reduction in cardiac output, and multisystem organ failure.[18]

Diagnostic testing should include complete blood count, blood, urine, and other cultures, electrolytes, enzymes, renal and liver function tests, arterial blood gases, coagulation panel, erythrocyte sedimentation rate, serology, chest roentgenogram, and electrocardiogram.[23, 24, 37] Other radiologic tests such as ultrasound, computed tomography, and nuclear scans may aid in diagnosis.[23, 37]

Monitoring of vital signs, temperature, and especially urine output should be established. Arterial and pulmonary artery catheters should be placed for management of volume status and to assess ventricular function.[9]

Treatment. Treatment is aimed at the primary diagnosis along with general measures to support circulation, respiration, and renal function.[9, 18, 52]

Antibiotics should be selected to cover the most likely organism, later refined on the basis of cultures and sensitivities, and the maximum intravenous doses given. If the site is uncertain, enteric gram-negative rods, anaerobes, and gram-positive cocci should be covered.

Mechanical ventilation should be instituted to support respiration, with careful titration of forced inspiration of oxygen and positive end-expiratory pressure.

Volume status should be carefully monitored with appropriate fluid management to maintain cardiac output and oxygen delivery and to improve the blood pressure. Blood, crystalloids, or colloids should be used as indicated.

Sodium bicarbonate may be given to treat metabolic acidosis.

Inotropic agents may be required for cardiovascular support. Their use is controversial. Dopamine is the agent of choice because of its effectiveness in increasing cardiac output and enhancement of renal and mesenteric blood flow. Low-dose dopamine (less than 4 μg/kg/minute) may preserve urinary output. Digitalis may be required to treat tachycardia and congestive heart failure.

Surgical exploration may be necessary if there is persistent evidence of a focus of infection.

Drug Reactions

Etiology. Drugs can interfere with central or peripheral physiologic mechanisms controlling body temperature and alter the balance between heat gain and loss.[19] Drug interactions with the thermoregulatory mechanism are complex and depend on ambient conditions and the physiologic state of the individual.[19] Certain drugs have hormonal effects whereas others affect energy transfer. Commonly used drugs that may have thermal effects and produce fever are antihistamines, barbiturates, belladonna, cimetidine, antihypertensives, antiarrhythmics, and antibiotics.[19] Anesthetic agents, cement, chymopapain, radio-opaque dyes, and colloids may also produce allergic reactions associated with fever.[41, 42]

Belladonna Drugs. Toxic effects of atropine or scopolamine may result from exposure to over-the-counter medications, eye drops, or parenteral administration. Dry mucous membranes, dilated pupils, flushed skin, tachycardia, fever, restlessness, excitement, and incoordination are evidence of autonomic dysfunction, characterized by parasympathetic suppression. Treatment is with physostigmine, sedation, and cooling measures.[54]

Allergic Reactions. Some drug reactions, depending on the target organ, may manifest as allergic reactions, with fever, urticaria, hypotension, laryngeal edema, or bronchospasm.[28] These types of reactions are mediated by histamine, seratonin, kinens, and other vasoactive substances. Anaphylaxis is a reaction involving multiple target organs. Treatment is geared to the cardiovascular, pulmonary, and cutaneous symptoms. Maintenance of airway and oxygenation, volume expansion, antihistamines, aminophylline, and corticosteroids may be required.[28] Epinephrine may be given intravenously for hypotension or for cardiovascular collapse.[27]

Recreational Drugs. The after effects of drug abuse may also appear in the PACU. Due to a changing patient population and trauma, which is often associated with the use of recreational drugs,[10] we are frequently faced with the management of conditions resulting from drug abuse in conjunction with surgery and anesthesia.

Psychedelic drugs of concern include hallucinogens (LSD), stimulants (amphetamines), deliriants (anticholinergics, cocaine), and euphoriants (hashish, marijuana).[10] These drugs are central nervous system, cardiovascular, and respiratory stimulants.[10, 29] They affect the central nervous system heat-regulating mechanisms, thereby increasing temperature and promoting hypermetabolism. Their primary effect is to block the reuptake of catechols.[10, 29]

An adrenergic storm is created by the release of catechols from nerve terminals in the central and peripheral nervous systems. Marked hyperpyrexia is associated particularly with cocaine, which has a vasoconstrictive effect and increases muscle activity.[29]

Acute physiologic effects related to sympathetic nervous system discharge in addition to hyperpyrexia are hypertension, tachycardia, flushing, sweating, muscle cramps, tremors, and vomiting.[10, 52] Anxiety, paranoia, hallucinations, seizures, and cerebral hemorrhage may occur. Uncontrolled hypertension may produce pulmonary or cerebral edema or hemorrhage.[29, 52] Coronary artery vasospasm, myocardial infarction, and fatal ventricular arrhythmias are common.[52]

Treatment is with chlordiazepoxide, diazepam, or barbiturates for sedation.[29] For control of blood pressure, alpha and beta blockers such as propranolol, esmolol, and labetolol are used.[16] Diazoxide may be given for vasodilation.[10] Treatment of arrhythmias with lidocaine or other antiarrhythmics, metabolic acidosis with sodium bicarbonate, and management of airway, oxygenation, and hydration are important.[16] Cooling measures should also be undertaken.

Neuroleptic Malignant Syndrome. The features of neuroleptic malignant syndrome, which is induced by phenothiazines and other psychotropic drugs, closely resemble those of malignant hyperthermia. Signs and symptoms include rigidity, dyskinesia, fever, changes in mental status, and autonomic nervous system manifestations such as tachycardia, blood pressure instability, and diaphoresis. The effects may persist for days, during which dehydration and exhaustion may occur.[11] Neuroleptic malignant syndrome is caused by the alteration of central mechanisms: dopamine receptor inactivation versus disturbance of hypothalamic temperature regulation. Treatment is with dantrolene, as for malignant hyperthermia, or bromocriptine, a dopaminergic agonist.[11]

Reaction to Blood and Blood Products

Etiology and Diagnosis. Febrile reactions may occur to pyrogens, aggregates, cell debris, and endotoxins associated with blood administration.[15, 28] Patients may react to major or minor group incompatibilities or develop thrombocyte and leukocyte reactions. Allergic manifestations may resemble drug reactions. If a transfusion reaction has occurred the patient may experience fever, chills, burning along the vein, flushing, urticaria, headache, myalgias, and arthralgias.[15, 28] More severe hemolytic transfusion reactions to ABO incompatibilities may be manifested by hypotension, tachycardia, chest or flank pain, respiratory distress, bronchospasm, bleeding, hemoglobinuria, and renal failure.[15, 28]

Treatment. Blood transfusions should be discontinued immediately and the blood should be returned to the blood bank. A blood sample from the patient should be sent for repeat cross match. A direct antiglobulin test will distinguish between a hemolytic and nonhemolytic transfusion reaction. Culture, coagulation studies, and urine test for hemoglobin should be obtained. Symptomatic treatment with antipyretics and antihistamines is indicated. Circulatory and renal support with mannitol, fluids, and alkalinization of the urine may be required if the reaction is severe.[15, 28]

Central Nervous System Catastrophe

Hemorrhage, trauma, or tumor may result in loss of temperature regulation producing hyperthermia, posturing, convulsion, or coma.[48] The patient may fail to awaken from surgery and anesthesia. There may be automonic nervous system dysfunction and bradycardia due to increased intracranial pressure.[48] CPK may be elevated. Neurologic workup and treatment measures designed to decrease intracranial pressure should be undertaken.

Vomiting and Aspiration

Aspiration may occur silently in the operating room or in the recovery room while a patient is still anesthetized and protective airway reflexes are absent. Symptoms include respiratory insufficiency, cyanosis, tachycardia, bronchospasm, fever, and sometimes hypotension.[28] The picture depends on the amount and the acidity of the aspirate. If the pH is less then 2.5, the aspiration is more serious. Arterial blood gases demonstrating hypoxemia and evidence of shunting, and chest roentgenograms showing an infiltrate, consolidation, or pulmonary edema confirm the diagnosis.[28]

The use of antibiotics and steroids does not affect outcome. A sputum culture should be obtained. If aspiration is severe, mechanical ventilation may be required, including suctioning, continuous positive airway pressure or positive end-expiratory pressure to counteract atelectasis and edema. Bronchodilators may also be useful.[28]

Exogenous Hyperthermia

In the operating room, patients may be smothered with occlusive drapes and plastic, preventing heat loss. This problem can be quickly resolved on admission to the PACU by removal of clothing and coverings. It may be exacerbated in a young child who has also received atropine.[19]

DIFFERENTIAL DIAGNOSIS

Distinguishing among those entities that are most likely to produce hyperthermia in the PACU is often difficult.

Malignant hyperthermia, thyroid storm, pheochromocytoma, and sepsis are similar in that they present dramatically, are triggered by stresses such as anesthesia and surgery, display nonspecific signs and symptoms (hyperthermia, tachycardia, tachyarrhythmias), are hypermetabolic states, are fueled by catecholamines, manifest compensatory mechanisms for the dissipation of heat, and require nearly identical supportive measures, although their pharmacologic management differs.

An analysis of manifestations affecting specific organ systems may permit differentiation (Tables 3, 4, and 5).

SUMMARY

The challenge of the PACU is provided by unexpected events, which require immediate action by the anesthesiologist. Hyperther-

Table 3. *Differential Diagnosis of Hyperthermia in the PACU: Signs and Symptoms*

MALIGNANT HYPERTHERMIA	THYROID STORM	PHEOCHROMOCYTOMA	SEPSIS
Fever	Fever	Fever	Fever
Tachycardia	Tachycardia	Tachycardia	Tachycardia
Atrial/ventricular arrhythmias	Atrial arrhythmias	Ventricular arrhythmias	
Blood pressure lability	Hypotension	Hypertension	Hypotension
Tachypnea		Dyspnea	Hyperventilation
Cyanosis, mottling	Flushing	Cyanosis	Flushing
Diaphoresis	Diaphoresis	Diaphoresis	
	Nausea, vomiting	Nausea, vomiting	GI bleeding
Rigidity	Agitation	Anxiety, headache	Confusion

Abbreviations: GI = gastrointestinal.

Table 4. *Differential Diagnosis of Hyperthermia in the PACU: Results of Laboratory Tests*

MALIGNANT HYPERTHERMIA	THYROID STORM	PHEOCHROMOCYTOMA	SEPSIS
Acidosis		Acidosis	Acidosis
Hypercarbia			Hypocarbia
Hypoxemia			Hypoxemia
Hyperkalemia	Hyperglycemia	Hyperglycemia	Electrolyte imbalance
Azotemia		Azotemia	Azotemia
Coagulopathy		Coagulopathy	Coagulopathy
			Erythrocytosis, leukocytosis
Myoglobin, elevated CPK	Elevated thyroid hormones	Elevated urinary/ plasma cathechols	Positive cultures

Table 5. *Treatment of Hyperthermia in the PACU**

MALIGNANT HYPERTHERMIA	THYROID STORM	PHEOCHROMOCYTOMA	SEPSIS
Dantrolene	Propylthiouracil Iodide	Phentolamine Phenoxybenzamine Propranolol Nitroprusside	Antibiotics
Antiarrhythmics	Steroids	Antiarrhythmics	Bicarbonate
Bicarbonate	Digitalis	Bicarbonate	Dopamine
Diuretics	Diuretics	Steroids	

* Primary and secondary treatment modalities. Supportive therapy with fluids, oxygen, and cooling measures are applicable to all hyperthermic syndromes.

mia is one such event. Because of its nonspecific nature and the many possible underlying etiologies, one must be an astute diagnostician to determine its correct cause. The responsible physician must utilize appropriate monitoring devices to aid in diagnosis and management. The necessary drugs, equipment, and personnel must be available in the PACU to handle a crisis. Since postoperative hyperthermia creates additional demands on the cardiovascular and respiratory systems during a period of diminished reserve, it is imperative that aggressive treatment be undertaken.

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Problem Management in the Ambulatory Surgery Postanesthesia Care Unit

*Samuel M. Parnass, MD**

The past 10 years have been characterized by a dramatic shift in surgical procedures to the outpatient setting. Outpatient anesthesia now accounts for over 30% of surgery at Rush-Presbyterian-St. Luke's Medical Center in Chicago, a tertiary referral center, and approaches 50% in many community hospitals.

It is no longer appropriate to restrict ambulatory procedures to only American Society of Anesthesiologists (ASA) physical status I or II patients. The ASA physical status III patient is acceptable if the patient's systemic disease is medically stable, and if the anesthesiologist assesses the patient's acceptability for ambulatory surgery through preoperative evaluation. The list of patients who are inappropriate for outpatient surgery is not very long though it is not without controversy (Table 1). More important to remember is that each case must be individualized, with the patient history, surgical procedure, and anesthetic technique all factored into the decision regarding the appropriateness of outpatient surgery for a specific patient.

PATIENT SELECTION

The patient is scheduled by the surgeon who in turn is responsible for the history and physical examination that must be provided for each patient according to Joint Commission on Accreditation of Health Care Organizations (JCAH) requirements. Simple screening questions have been shown to correlate with a consensus of fitness

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Table 1. *The Inappropriate Patient for Ambulatory Surgery*

1. Premature infant less than 45 weeks post conceptual age
2. Infants with severe respiratory distress syndrome, bronchopulmonary dysplasia, or family history of sudden infant deaths, less than 6 months old
3. Patients susceptible to malignant hyperthermia
4. Patients with uncontrolled seizure activity
5. Medically unstable ASA status 3 (or 4) patients
6. Morbidly obese patient with systemic disease
7. Acute substance abuse
8. Patient who is uncooperative or who has no responsible adult available

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for anesthesia (Table 2). These questions can easily be answered by patients and serve as a screening or "red-flag" as to which patients need further work-up. In general, it is preferable for some preoperative screening to be performed prior to the day of surgery by the anesthesiologist either through chart reviews, telephone interviews, or direct history and physical examination. In many centers, however, the patients are first seen by the anesthesiologist the day of the surgery, and this is usually adequate for the healthy ASA class I or II patient. There is a small risk, however, of these patients being cancelled or delayed should abnormalities be found the day of surgery. On the other hand, all class III patients should be reviewed prior to the day of surgery to make sure they are ambulatory candidates and that their medical problems are optimized and stable.

Outpatient centers need to develop guidelines as to what their minimal laboratory test requirements are. At Rush-Presbyterian-St. Luke's Medical Center we have separate requirements for class I and II versus class III patients. Table 3 lists the current requirements. We

Table 2. *Problems in Anesthesia: Questions to Evaluate Fitness for Anesthesia*

1. Do you feel sick?
2. Have you had any serious illnesses in the past?
3. Do you get more short of breath on exertion than others of your age?
4. Do you have a cough?
5. Do you have a wheeze?
6. Do you have any chest pain on exertion?
7. Do you have any ankle swelling?
8. Have you taken any medications in the past 3 months?
9. Have you any allergies?
10. Have you had an anesthetic in the past 2 months?
11. Have you or your relatives had any problems with anesthesia?

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Table 3. *Rush-Presbyterian-St. Luke's Medical Center, Chicago, Illinois, Department of Anesthesiology Policies and Procedures*

1. Routine admission for outpatient surgery is limited to those patients classified as class I and II by the American Society of Anesthesiologists Patients Status Classification. Class III patients may be admitted if they are medically stable and have anesthesia clearance
2. The minimal requirements for an adult outpatient surgery patient (class I or II) having surgery under general anesthesia are:
   History and physical examination
   Hematocrit
   Sickle preparation as indicated
   Chest roentgenogram for patients over 60 years of age
   EKG for male patients over 40 years of age and for female patients over 60 years of age
   Valid consent
3. The minimal requirements for any adult class III ambulatory surgery patient having surgery under general anesthesia, local/general, or major regional blocks are:
   History and physical examination
   CBC
   Urinalysis
   SMA-6 with glucose
   Chest roentgenogram
   EKG
   Sickle cell preparation as indicated
   Valid consent
4. For class I and II patients, lab data, EKG, and chest roentgenogram are accepted up to 6 months before surgery if there has been no intervening illness or change in the clinical status
5. For class III patients, laboratory data, EKG, and chest roentgenogram are accepted up to 4 weeks before surgery if there has been no intervening illness or changes in the clinical status

Abbreviations: EKG = electrocardiogram; CBC = complete blood count.

review the results at least 24 hours prior to surgery, with class III patients being scheduled for an anesthesia preoperative assessment.

It is the decision of the individual anesthesiologist as to what range of laboratory abnormalities are acceptable based on the specific clinical situation. However, the old adages that the hemoglobin must be greater than 10 g/mL or the potassium greater than 3.5 mEq/L are no longer applicable given the current climate regarding transfusion-associated disease and current opinion that mild hypokalemia is safe for patients undergoing general anesthesia.

ANESTHETIC TECHNIQUES

Regional Anesthesia

Many of the common problems associated with general anesthesia, such as oversedation, nausea and vomiting, and residual pain, are generally considered benign for inpatients; however, they are very significant in the outpatient setting. They delay or prevent discharge, disrupt the smooth functioning of ambulatory surgery units, and aggravate patients and their families who are eager to return home. Regional anesthesia, on the other hand, when properly selected and performed, can avoid these problems and help make the outpatient experience significantly better. When performed with light sedation, patient anxiety can be controlled without delaying discharge time from oversedation. There is a lower incidence of nausea and vomiting after regional anesthesia when compared with general anesthesia, as well as a lower incidence of unanticipated hospital admissions. Postoperative analgesia, either from residual block (i.e., axillary block) or supplementation with local infiltration, can also serve to effect early ambulation and avoid admission for intractable pain. The elderly in particular, being more sensitive to intravenous agents and more susceptible to prolonged sedation, may benefit tremendously from regional anesthesia.

Successful regional anesthesia depends on a number of important factors. Proper patient selection is very important, as well as education regarding the technique (specifically, the performance of the block), its residual effects, and side effects. Similarly, there must be full support from the surgeon, who is usually the first person to discuss anesthesia possibilities with the patient. The surgeon and nursing staff must be aware of the extra time that may be needed for certain techniques, and realize the payoff that this investment will have later. A holding area where blocks can be performed will help facilitate patient flow and avoid delays, keeping rapid turnover of the surgical schedule possible. Patient cooperation is extremely important, and the highly anxious patient is not a good candidate for regional anesthesia. If too much sedation is used, discharge time will be correspondingly prolonged.

Proper choice of local anesthetic agent is also very important. In contrast to the inpatient in whom long-acting agents are advantageous in providing postoperative analgesia, the outpatient needs to be discharged in a timely manner. Therefore, shorter-acting agents are best used for spinal or epidural blocks, with reliance on local wound infiltration, analgesics, or both to provide adequate postoperative pain relief. With the use of crutches for lower extremity blocks and slings for upper-extremity blocks, however, long-acting agents can be used in the properly selected patient, after careful explanation about the need to avoid any external trauma to the affected limb.

Regional anesthesia does not eliminate the potential for aspiration, nor does it eliminate the need for adequate preoperative evaluation. Ambulatory patients are at risk for significant pulmonary complications from aspiration of gastric contents and must take nothing by mouth. Adequate preoperative evaluation and monitoring are also important because as many as 5% of patients with "just local" anesthesia (i.e., cataract surgery) have been shown to generate emergency calls for an anesthesiologist. More than 50% of patients undergoing "local" or "local/sedation" anesthesia have significant variations of blood pressure and heart rate, and many of these patients have been shown to have oxygen saturations less than 85% at some point during the procedure. This demonstrates the importance of close monitoring in these groups of patients.

There are, of course, problems with regional anesthesia that may be encountered in the outpatient setting that practitioners need to be aware of and know how to handle. First and foremost is patient anxiety, which can usually be addressed with proper education, a positive approach, and light sedation. The overly anxious patient, however, is clearly a poor candidate and should be avoided. The extra time required to perform certain blocks can be a significant drawback in busy outpatient centers. Separate anesthesia-induction areas can overcome this problem, or else the choice of technique should be limited to those that can be performed rapidly in relation to the expected length of the case. The time required for the local anesthetic to take effect is a factor and can be hastened by the appropriate use of rapidly acting drugs or by modifying them with alkalinization. One milliliter of $NaHCO_3$ added per 10 mL of 2% lidocaine with epinephrine significantly shortens the onset time for epidural block. Much less bicarbonate is needed for bupivacaine, where only 0.1 mL of $NaHCO_3$ added per 10 mL of 0.5% bupivacaine has been shown to significantly shorten onset time for axillary blocks (range of 20 to 32 minutes to 7 to 14 minutes) with a much longer duration of action.

The problem of residual numbness following regional block of an extremity has raised concern; however, no documented problems have been reported after either arm or foot blocks when careful instructions are given to patient and family about the need to protect the limb from external injury. Furthermore, the extra analgesia provided more than compensates for the extra care needed.

Urinary retention following spinal or epidural blockade is another problem occasionally encountered in the PACU; however, it is not a very significant problem when long-acting agents such as bupivacaine and tetracaine are avoided. Hypotension, from residual sympathectomy, is not a significant problem as long as ambulation is not attempted prior to documentation of full return of perianal sensation, and proprioception. In fact, there are fewer unanticipated hospital admissions from urinary retention and residual sympathectomy than from intractable nausea and vomiting after general anesthesia.

Post-dural puncture headache (PDPH) following either spinal or epidural anesthesia can be a significant problem. Although the risk does not increase with ambulation, the onset is hastened. The incidence of PDPH in young outpatients varies among reports from a low of 2% to a high of almost 40%, even with a 25-gauge needle. To avoid PDPH, epidural anesthesia has been recommended for young patients and, although the risk of dural puncture is still present, it is markedly reduced. Patients who cannot be adequately followed up, however, should not have either technique done.

Should a dural puncture occur, one can perform a prophylactic epidural blood patch. Although controversy exists regarding its efficacy, successful results have been reported, including the performance of a patch through the epidural catheter an interspace above the dural puncture site. Alternatively, conservative therapy, and counsel for bed rest, hydration, and analgesics, can be given over the telephone. Caffeine can be used as an effective method of therapy by the intravenous route and, in our experience, has provided relief in outpatients when prescribed orally over the telephone in the form of coffee, the caffeine-rich soft drink "Jolt," or over-the-counter caffeine preparations (i.e., NoDoze). If the headache persists, the patient should be brought back for either intravenous caffeine or an epidural blood patch. A recent protocol for intravenous caffeine included 2 L of intravenous fluid, each with 500 mg of caffeine sodium benzoate given at a rate of 1 L/hour. A second course of caffeine is given if needed after a 4-hour rest period. This protocol has been successful in 75% of cases, and patients refractory to this protocol can receive an epidural blood patch. Outpatient epidural blood patches can be performed simply and successfully without even the need for intravenous infusion, and patients can be discharged after a short period of observation.

Certain specific regional blocks may be inappropriate for procedures in which rapid discharge and minimal follow-up are routine. For example, sciatic-femoral block may prolong recovery time because of loss of motor strength and coordination in the blocked leg, whereas a supraclavicular brachial plexus block may be contraindicated because of the potential for pneumothorax.

Intravenous regional anesthesia (Bier block) is particularly well suited for orthopedic outpatient procedures because of the ease and simplicity of performing the block, as well as its rapid disappearance following release of the tourniquet. When properly performed, this technique provides excellent analgesia, with adequate muscle relaxation. Although most studies on intravenous regional anesthesia deal with surgery of the upper extremities, satisfactory surgical analgesia can be obtained for short procedures of the foot. The tourniquet is placed below the knee, avoiding pressure over the superficial peroneal nerve. Lidocaine is most commonly used (3 mg/kg or 40 mL of 0.5% solution) for the upper extremity, whereas 75 to 100 mL of a 0.25 to 0.35% lidocaine solution has been suggested for lower-extremity surgery.

General Anesthesia

The aim of general anesthesia is, in many respects, similar to that of regional anesthesia previously discussed: induction and maintenance of a smooth anesthetic with rapid safe recovery, and minimal postoperative side effects. Particular attention must be paid to avoid postoperative psychomotor and cognitive dysfunction, postoperative pain, and postoperative nausea and vomiting when choosing general anesthetic techniques. These are the problems that will have to be dealt with in the PACU and will be much easier to deal with if they can be moderated or avoided completely. Therefore, this article first discusses what anesthetic techniques or agents are ideal for outpatients, and then discusses the specific problems of pain control and nausea and vomiting in the PACU.

Premedication. The routine use of premedication in outpatients is often avoided because of concern about prolonged postoperative sedation and recovery times. Furthermore, many centers do not have an area available where the patient can be monitored once the premedication is given. The use of potent amnestic agents such as midazolam may cause patients to not remember having actually seen their surgeon or anesthesiologist the day of surgery. Patients also may not remember getting postoperative instructions or receiving answers to their questions. There are, of course, patients who are very anxious and in whom some premedication would be appropriate. Attention to agents that are relatively short acting and avoidance of very large doses will allow appropriate and timely discharge. In general, the administration of small intravenous doses of short-acting narcotics such as fentanyl, or sedatives such as midazolam, can be used as part of a balanced anesthetic technique in conjunction with premedication. This will circumvent the problem of preoperative monitoring for patients medicated prior to arrival in the unit or in units where no monitored area is available. In fact, fentanyl, when used as an intravenous premedication or as an adjunct to balanced anesthesia in a dose of 1 to 2 μg/kg, has been shown to decrease recovery time as a result of the narcotic's ability to decrease anesthetic requirements. Furthermore, because postoperative pain has been well established as a factor in postoperative nausea, the use of small doses of intravenous narcotics may reduce the amount of postoperative pain and, thereby, nausea and vomiting, which can increase the time needed for recovery.

Other agents have traditionally been used for premedication, such as anticholinergics to block vagal reflexes, antacids and histamine (H_2) receptor blockers to increase the pH of gastric contents, and gastrokinetic agents to reduce the volume of gastric contents. These may be used in outpatients when specifically indicated. For example, patients with a history of peptic ulcer, hiatal hernia, obesity, or heartburn may benefit from preoperative medication with antacids, H_2 blockers, or gastrokinetic drugs. The routine administration of antacids or H_2 blockers is probably not indicated for all outpatients: the risk of aspiration is small to nonexistent based on large outcome studies, and there are no data available that support such a practice.

Intravenous Anesthetics. Intravenous anesthetic agents are most commonly used for induction of general anesthesia, although they are also used for maintenance of anesthesia, usually as adjuvants to nitrous oxide. Methohexital has the shortest recovery time when compared with thiopental and etomidate. Most recent work, however, focuses on the use of propofol, a new intravenous anesthetic agent. Propofol is particularly well suited for outpatient anesthesia because of its purported superior quality and speed of recovery. This more rapid recovery, along with a decreased propensity for nausea and vomiting, may help prevent a significant portion of the problems currently seen in the PACU. Propofol, like thiopental, causes slightly greater cardiorespiratory depression in comparison with methohexitone when used as an induction agent, but the speed and quality of recovery are superior. When compared with thiopental, propofol was found to have a significantly greater number of "excellent inductions," with more rapid recovery, when used for induction of anesthesia. Recovery times for awakening, orientation, and ambulation are consistently shorter with propofol, compared with methohexital, when used as adjuvants to nitrous oxide anesthesia for outpatients. Propofol has been associated with fewer side effects such as hiccupping, nausea, and vomiting in both the intraoperative and postoperative settings. Common side effects from propofol include cardiovascular depression similar to that produced by thiopental, and pain on injection depending on the site of administration (greatest with dorsal hand veins).

Intravenous Analgesics. The most common intravenous analgesic agents being used in outpatient anesthesia are fentanyl and alfentanil. Both drugs are relatively short acting in comparison with morphine and are most frequently administered as part of a balanced anesthetic technique. Narcotic techniques in combination with nitrous oxide have been used for outpatient surgery; however, an unacceptably high incidence of nausea and vomiting has made these techniques less favorable.

Although even small pre-induction doses of intravenous narcotic agents have been shown to increase the incidence of postoperative nausea and vomiting, they are beneficial because they reduce anesthetic requirements. They also decrease the need for postoperative pain control, which will decrease recovery time, as well as decrease the incidence of nausea from pain alone.

Inhalational Agents. There is currently insufficient evidence to strongly suggest any one of the currently available inhalational agents over another for outpatient surgery. Although enflurane has been shown to have shorter recovery times when compared with halothane in procedures that were longer than 90 minutes, recovery was significantly faster with isoflurane. This suggests that isoflurane may be the inhalational agent of choice for outpatients in procedures lasting longer than 90 minutes.

Nitrous oxide is the most common adjunct anesthetic for both inhalational and intravenous anesthesia techniques. Considerable controversy exists regarding the role of nitrous oxide in the production of postoperative nausea and vomiting. Despite this potential drawback, however, nitrous oxide's pleasant odor and characteristically rapid induction and emergence make it an important adjunct agent for both inhalational and intravenous anesthesia in the outpatient.

Muscle Relaxants. The rational use of muscle relaxants in the outpatient setting is important in avoiding postoperative problems of weakness, myalgia, or a phenomenon such as "recurarization." This is because outpatient procedures are generally short in nature and no ideal short-acting nondepolarizing muscle relaxant is currently available in the United States. Many outpatient procedures, of course, will not require muscle relaxation for the surgical procedure, and many cases can be performed by face mask and will not require endotracheal intubation. When endotracheal intubation will be used, the most common muscle relaxant to facilitate intubation is succinylcholine. Although succinylcholine is an ideal drug in the outpatient setting because of its rapid onset and ultrashort-acting duration, its numerous side effects, particularly myalgia, can be quite significant for outpatients. Myalgia after succinylcholine can occur up to 4 days postoperatively and may be more severe than the surgical procedure itself. The use of a defasciculant may decrease the incidence of myalgia, and the preferred agents are those with significant prejunctional effects, such as gallamine and tubocurarine. Myalgia, however, can occur even in the absence of fasciculations, as well as in patients receiving nondepolarizing muscle relaxants only.

When muscle relaxation is needed for the surgical procedure in cases less than 20 minutes long, the only available option right now is a succinylcholine infusion. There are no currently approved short-acting nondepolarizing muscle relaxants in the United States. Mivacurium (BW B1090U) has been under clinical investigation since 1985. Structurally, mivacurium is similar to atracurium and also undergoes ester hydrolysis, though there is no Hofmann elimination. Mivacurium has the same weak histamine-releasing properties as atracurium and may produce hypotension, tachycardia, and a skin flush on induction. Intubating doses of 0.2 to 0.25 mg/kg have an onset time of 2.5 minutes, with a 95% recovery in only 30 minutes. This makes the intubating doses of mivacurium about twice as long as succinylcholine and half as long as atracurium and vecuronium. This is close to an almost ideal muscle relaxant for short outpatient procedures when needed for either surgical relaxation or intubation. Mivacurium will rarely need to be reversed with anticholinesterases although 5% recovery is seen at 13 minutes with a dose of 0.15 mg/kg and can be reversed at that point.

For procedures lasting longer than 30 minutes, the intermediate-acting nondepolarizers, atracurium and vecuronium, are valuable new clinical options. The duration of action of both drugs is similar, although when used in intubating doses, the duration of action of 50 to 70 minutes may be too long for short ambulatory cases. A combination of both atracurium (0.125 mg/kg) and vecuronium (0.025 mg/kg) has been shown to produce neuromuscular blockade with onset times similar to intubating doses of either drug alone, yet with faster recovery. The combination was found to have a recovery to 25% in 32 minutes and a recovery to 90% in 48 minutes. This combination could have significant advantages in an outpatient setting until mivacurium becomes available.

PAIN MANAGEMENT IN THE POSTANESTHESIA CARE UNIT

Adequate management of postoperative pain, whether preventive or as treatment, is one of the most important factors in successful outpatient surgery. Pain is the most common surgically related reason for unexpected hospital admissions and is a major determining factor in deciding when patients are ready for discharge. Patients' perceptions of the quality of their overall ambulatory surgery experience will also be greatly enhanced by the rapid and effective control of postoperative pain.

The cornerstones of pain management in ambulatory surgery include the following: the use of potent intravenous opioid analgesics in the preoperative and intraoperative periods; the use of local anesthetic agents as either supplements to general anesthesia or at the end of surgery for postoperative analgesia alone (given either as a regional block or via local infiltration); and, finally, the use of potent oral analgesics, both opioid and nonsteroidal anti-inflammatory agents, for the relief of pain at home.

The use of opioid analgesics in combination with general anesthesia has been shown to enhance recovery times by decreasing postoperative pain. Because the use of narcotic analgesics, however, is also associated with nausea and vomiting, one must use them judiciously and in adjunctive doses to get the maximum benefit with the least side effects. When used as premedicants, they have been shown to decrease patient anxiety, reduce anesthetic requirements, and provide pain relief in the postoperative period. Fentanyl (1 to 2 μg/kg), sufentanil (0.1 to 0.25 μg/kg), or alfentanil (7.5 to 15 μg/kg) are drugs that have all been used successfully, with significantly reduced pain postoperatively, yet without an increase in nausea or vomiting. Patients who receive inhalational anesthesia without narcotics, have a significantly higher incidence of postoperative pain and a higher incidence of excitement during their recovery period, whereas patients who receive an opioid intraoperatively recover sooner and spend less time in the PACU. There are numerous intravenous opioid analgesics that can be used for ambulatory surgery and no ideal agent has yet been determined. Although it has been suggested that long-acting drugs such as morphine and meperidine may have too long an action for outpatient use, data comparing meperidine (1 to 1.5 mg/kg) with fentanyl (2 μg/kg) have not shown any differences in recovery or patient discharge times. Nalbuphine is generally thought to be as effective as morphine when used at a dose of 0.25 mg/kg. In comparison with fentanyl (2 mg/kg), nalbuphine patients have been shown to have significantly lower pain scores at 1 hour, requiring significantly less postoperative analgesia. Butorphanol (20 μg/kg) is as effective as fentanyl (2.0 μg/kg) as a supplement to balanced general anesthesia, although patients receiving butorphanol (40 μg/kg) are generally more drowsy. The incidence of nausea and vomiting is similar for both drugs.

The use of local anesthetics has been successful in preventing and treating pain in the PACU. Infiltration of the ilioinguinal and iliohypogastric nerves with bupivacaine will decrease pain and vomiting during the recovery period, as well as significantly decrease anesthetic and analgesic requirements, with a more rapid return to normal activity. After laproscopic tubal ligations, infiltration of the mesosalpinx with 0.5% bupivacaine in the area of the fallopian ring placement, as well as topical 1% etidocaine, has been shown to significantly decrease pain in the PACU. A subcutaneous ring block of the penis with 0.25% bupivacaine will decrease PACU analgesic requirements after circumcision in children. The application of topical lidocaine jelly alone is a simple maneuver that is as effective as dorsal nerve block and parenteral morphine for outpatient circumcisions. The use of intra-articular bupivacaine after arthroscopic meniscectomy will significantly reduce the patient's assessment of pain, as well as significantly reduce the amount of supplemental morphine required postoperatively. Caudal block supplementation can be used to prevent postoperative surgical pain for pediatric circumcision, correction of hypospadius, and orchiopexy. Caudal analgesia has been shown to be superior to systemic opioids with a more tranquil period, superior analgesia, and a lower incidence of vomiting.

NAUSEA AND VOMITING IN THE POSTANESTHESIA CARE UNIT

Postoperative nausea and vomiting is one of the major concerns that we face in the PACU. It is the most common complication reported in ambulatory surgery centers and is a common reason for unanticipated hospital admission, both for uncontrolled postoperative nausea and vomiting, and somnolence following treatment with potent antiemetics. In the outpatient setting, the problem of postoperative nausea and vomiting is most vexing because the consequences of delayed discharge times and the need for unanticipated hospitalization are more acutely felt. Notwithstanding a perfect surgical result and a well-delivered anesthetic, the ambulatory surgery experience can be viewed as a complete failure by outpatients who subsequently have uncontrolled postoperative nausea and vomiting or who need to be admitted.

Numerous factors have been associated with postoperative nausea and vomiting, some of which are controversial. An understanding of these factors (Table 4) will allow for the tailoring of an anesthetic that will minimize the incidence of postoperative nausea and vomiting, and, hopefully, prevent one of the major problems faced in the PACU. Knowing which patients and which surgical procedures are more prone to postoperative nausea and vomiting will help target these groups for aggressive prophylaxis.

Anesthetic Techniques

Anesthetic techniques such as the use of positive pressure ventilation by mask, often performed during induction of anesthesia to preoxygenate patients, may increase the incidence of nausea and vomiting. Positive pressure ventilation may inadvertently force air into the stomach, causing subsequent gastric distension and, thereby, postoperative nausea and vomiting. This may be even more significant in obese patients for whom ventilation by mask may be more difficult. The length of anesthetic exposure time has also been correlated with

Table 4. *Factors Associated with Postoperative Nausea and Vomiting*

1. Anesthetic techniques
2. Anesthetic agents
3. Gender
4. Age
5. Weight
6. Surgical procedure
7. Pain
8. Prior history of postoperative nausea and vomiting
9. History of motion sickness

an increased incidence of subsequent postoperative nausea and vomiting.

Anesthetic Agents

Certain anesthetic agents used for the induction and maintenance of anesthesia have been shown to influence the incidence of postoperative nausea and vomiting. Although there is controversy regarding some of these agents, it is clear that the use of a narcotic-based technique for the induction or maintenance of anesthesia increases the incidence of postoperative nausea and vomiting.

Nitrous oxide has classically been implicated as a major cause of postoperative nausea and vomiting, although this has recently been questioned and is currently controversial. The implication of nitrous oxide as a cause for postoperative nausea and vomiting has been based on the increased sympathetic activity of nitrous oxide, changes in middle ear pressure, and gastrointestinal distension from nitrous oxide. However, these theoretical observations have not been well documented, and recent evidence fails to support the association between nitrous oxide and postoperative nausea and vomiting.

There is no strong consensus regarding the different inhalational anesthetic agents. Enflurane has been suggested as the inhalation agent of choice with gynecologic outpatients as it appears to be associated with less nausea and vomiting; however, other studies have found no differences among the inhalational agents. Regional anesthesia, with the avoidance of potent inhalational or intravenous anesthetic agents, has been shown to be accompanied by significantly less postoperative nausea and vomiting.

Age, Gender, and Weight

The incidence of postoperative nausea and vomiting is correlated with younger age groups. Women are two to four times more likely to experience postoperative nausea and vomiting than men.

The issue of weight and obesity is somewhat controversial, but both have been implicated regarding postoperative nausea and vomiting. It had been thought that obese patients have an increased incidence of nausea and vomiting because obese patients sequester drugs in fat compartments, have slower metabolism, and prolong the release of anesthetics. However, recent studies have shown that body mass index is not associated with increased postoperative nausea and vomiting when one does not ventilate patients by mask prior to the induction of anesthesia. This is because because ventilating by mask increases the likelihood of gastrointestinal distension from air being forced into the stomach in obese patients, since they are generally more difficult to ventilate.

Type of Surgical Procedure

Whether the type of surgical procedure is an important predictive factor in the incidence of postoperative nausea and vomiting has been the subject of much debate; however, in the pediatric population it has been clear that strabismus and orchiopexy surgery have a significantly higher incidence of postoperative nausea and vomiting. Tonsillectomies and adenoidectomies have been similarly associated with an increased incidence of postoperative nausea and vomiting.

In the adult population, increased frequencies in postoperative nausea and vomiting have been reported in patients undergoing gastrointestinal procedures, as well as following otologic, opthalmic, and laparoscopic procedures. Patients with increased nausea and vomiting have a correspondingly extended recovery room stay by more than 50%. Prolonged recovery stays can also disrupt patient flow, and it may be prudent to establish daily scheduling principles, in which patients scheduled for procedures with higher incidences of nausea and vomiting and longer recovery room times would be scheduled early in the day. Additionally, it may be desirable to have a separate step-down recovery unit available for these patients so that patient flow will not disrupt the functioning or capacity of the ambulatory surgery unit.

Pain

The role of postoperative pain as an etiologic factor in postoperative nausea and vomiting has been well established. Many patients with inadequate pain relief and postoperative nausea have both symptoms relieved after receiving injections of potent intravenous analgesics. It is important, therefore, to remember that nausea often accompanies pain in the postoperative period, and it can be relieved in many cases when pain relief is achieved by the use of intravenous opiates.

Preoperative History

A good preoperative history is essential in outpatients who will be traveling home after their procedure. Patients who have a prior history of postoperative nausea and vomiting will be more likely to have postoperative nausea and vomiting with subsequent anesthetics. Similarly, patients with a history of motion sickness have a higher incidence of postoperative nausea and vomiting. A good preoperative history is important in identifying those patients who will be more prone to postoperative nausea and vomiting to enable us to target these patients for aggressive prophylaxis. The drive home from an outpatient unit may in itself stimulate post-PACU nausea and vomiting in patients who did not have any nausea or vomiting in the PACU.

Prophylaxis and Treatment of Postoperative Nausea and Vomiting

It is clear that the management of postoperative nausea and vomiting should ideally stem from a prophylactic rather than a therapeutic approach, particularly in those patients identified to be at risk. Such management serves to reduce the discomfort experienced by patients going through ambulatory surgery units. As these patients are generally less sedated, they will be more likely to remember incidents of nausea and vomiting when compared with their inpatient counterparts. Furthermore, the delay in discharge caused by nausea and vomiting tends to cause a further deterioration of the outpatient experience, as well as upset the unit's patient flow. Preventive therapy may minimize morbidity, increase and maintain patient comfort, and decrease the frustration often felt by nurses trying to manage the problem. But preventive therapy will not totally eliminate the incidence of postoperative nausea and vomiting, and timely intervention in the PACU is very important. It is advantageous for those involved in taking care of these patients, specifically PACU nurses, to have standing orders available for the treatment of both postoperative nausea and vomiting, and pain. This will avoid delays that are so common in trying to get the anesthesiologist or surgeon to give specific orders each time. Pre-printed order forms with strict guidelines will allow prompt treatment of both postoperative nausea and vomiting, and the pain that may be contributing to them.

There have been many different agents tried for the control of postoperative nausea and vomiting, and there is controversy regarding some of them. Common agents that have been used are listed in Table 5. The problem with most of these medications is that although they may be very efficacious in preventing or controlling postoperative nausea and vomiting, increases in dosages also increase the incidence of postoperative sedation. Although this may not be a significant problem in the inpatient population, it is an important problem for outpatients and can adversely affect discharge times.

The two most common agents used today include metoclopramide and droperidol. Droperidol is effective in a low-dose intravenous for-

Table 5. *Agents Used for Treatment of Postoperative Nausea and Vomiting*

1. Droperidol (Inapsine)
2. Metoclopramide (Reglan)
3. Benzquinamide (Emete-Con)
4. Prochlorperazine (Compazine)
5. Trimethobenzamide hydrochloride (Tigan)
6. Hydroxyzine (Vistaril)
7. Ephedrine sulfate
8. Dimenhydrinate (Dramamine)

mat without significantly affecting discharge times. Dosage recommendations range from 0.625 to 2.5 mg for the average adult (10 to 20 μg/kg), whereas dosage guidelines for children range from 25 to 75 μg/kg.

Metoclopramide has similarly been found efficacious for nausea and vomiting. Both metoclopramide and droperidol antagonize dopaminergic receptors and block stimulation of the medullary chemoreceptor trigger zones. However, only metoclopramide is a gastroprokinetic drug, which sensitizes the upper gastrointestinal tissues to the action of acetylcholine, thereby stimulating gastric motility. Metoclopramide also increases the resting tone of the lower esophageal sphincter, relaxes the pyloric sphincter and duodenal bulb during gastric contractions, and simultaneously increases peristalsis of the proximal small bowel. The net result is an accelerated gastric emptying time and shortened small bowel transit time. This can be important in the preinduction period to help eliminate gastric contents and prevent any chance of aspiration, while also helping to decrease the incidence of nausea and vomiting. Prior treatment with anticholinergic agents does not inhibit the gastroprokinetic actions of metoclopramide in normal patients as it does in obese patients, though the reasons for the difference are not clear. The duration of action of metoclopramide is approximately 2 hours, and if the drug is given preoperatively for a procedure that lasts 1 to 2 hours, there may not be enough drug left postoperatively. It is, therefore, advantageous to give a second dose of metoclopramide at the end of the procedure in order to get good postoperative nausea and vomiting prophylaxis. Higher doses than the average 10 mg for a 70-kg patient may be needed in certain populations, especially women. Of special interest is the fact that oncology patients routinely receive 50 mg metoclopramide per dose for nausea prophylaxis prior to receiving chemotherapeutic agents. We commonly give patients with a history of motion sickness or previous history of postoperative nausea and vomiting 20 mg metoclopramide preoperatively, and repeat the dose prior to extubation. One of the major advantages of metoclopramide is that there is little or no sedative effect.

There are side effects to both droperidol and metoclopramide that must be kept in mind. Droperidol may cause sedation and can also potentiate other central nervous system depressants commonly used in the postoperative period. Droperidol, as an alpha-blocker, may also cause hypotension and peripheral vascular dilatation. Caution should be used in patients who are hypovolemic, dizzy upon standing, or have low blood pressure. Droperidol may also cause an acute dysphoric reaction as well as extra-pyramidal symptoms, such as dystonia or oculogyric crisis. Metoclopramide is less likely to cause extra-pyramidal syptoms, although these have been reported. It must be remembered that metoclopramide with its gastroprokinetic actions is contraindicated with bowel obstruction or partial bowel obstructions, and both drugs should be avoided with Parkinson's disease. The treatment for extra-pyramidal reactions should they occur is benztropine, 1 to 2 mg (Cogentin), or diphenhydramine, 25 to 50 mg (Benadryl), intravenously.

Although the incidence of postoperative nausea and vomiting has been clearly shown to be decreased with droperidol and in many studies with metoclopramide, the incidence has not been completely reduced. Combination therapy, therefore, has been employed as a method of increasing the amount of antiemetic drug given without a concomitant increase in the incidence of sedation or side effects. The combination of droperidol and metoclopramide has been found to be more effective in preventing nausea and vomiting than droperidol alone, with concomitant shortening of ambulation and discharge times.

Other investigators have approached the problem of postoperative nausea and vomiting from the perspective of motion sickness. It has been well documented that a history of motion sickness is a strong predictive factor in postoperative nausea and vomiting. Dimenhydrinate (Dramamine), a commonly used antimotion sickness drug was found to significantly decrease the incidence of nausea (8%) when compared with both droperidol (21%) and placebo (34%). Dimenhydrinate is an antihistamine and its antimotion sickness effect is thought to be a combination of a primary H_1-blocking effect and central anticholinergic action.

Ephedrine has also been studied for the prevention of postoperative nausea and vomiting in outpatients. The prophylactic use of ephedrine (0.5 mg/kg intramuscularly after extubation) was found to be as effective as droperidol in reducing the incidence of nausea and late vomiting, with significantly less postoperative sedation in the ephedrine group. There were no significant differences in mean arterial blood pressure among the groups. Ephedrine has been used for the prevention of motion sickness by the National Aeronautics and Space Administration (NASA) and is commonly used to treat nausea and vomiting following hypotension after spinal or epidural anesthesia. Its mechanism for motion sickness is unknown, but following spinal and epidural anesthesia, it undoubtedly reverses the hypotension of the induced sympathectomy through its alpha-agonist properties. Similarly, many patients are volume depleted postanesthesia and may have residual vasodilation from potent inhalational anesthetics. This is typically manifested by dizziness upon standing and often accompanied by nausea and vomiting. These patients may particularly benefit from treatment with ephedrine in the PACU.

Persistent postopertive nausea and vomiting may require repeated doses of antiemetics in the PACU. Standing orders should be available for prompt and rapid institution of such therapy and careful attention must be given to pain control and the patient's volume status. It must be remembered that intravenous fluids should be increased to account for fluid losses and should be maintained until oral fluids are tolerated. Fluid losses should be replaced with lactated Ringer's or normal saline solution and should not be replaced with 5% dextrose in water alone because it will not remain in the intravascular compartment.

It is essential, furthermore, that good communication be conducted with the patient and his or her family. Patients should be warned prior to the procedure, during the preoperative interview, that nausea and vomiting are common side effects of anesthesia and surgery. They will then be able to tolerate it with less anxiety by expecting it as a possibility, and be less prone to feel that their experience has been a failure. Although it is rare, there will be the occasional patient in whom postoperative nausea and vomiting is refractory to all efforts to control it, and these patients will need to be admitted overnight. A review of unanticipated admissions over a 6-month period at the Ambulatory Surgical Unit of Rush-Presbyterian-St. Luke's Medical Center found that 10% of such admissions were for uncontrolled postoperative nausea and vomiting, with an overall incidence of 0.4%. Patients with persistent nausea and vomiting should be aggressively treated with maximal doses of antiemetics, as it is preferable to admit a patient for sedation with controlled nausea and vomiting, than it is to admit him unsedated and with uncontrolled nausea and vomiting.

Discharge Criteria

There are varying stages of recovery that patients go through after surgery and anesthesia. Sophisticated tests that can measure subtle psychomotor differences have been developed to measure these stages, but they are usually too complex and time-consuming to be used on a daily basis. Practical discharge criteria, therefore, have to be devised and implemented by each ambulatory surgery unit. In general, the criteria need to define the point at which the patient is "home ready." These criteria do not mean that the patient is street-fit or ready to drive (Table 6).

Most centers generally follow a sequence that includes a stage I PACU where the patient is kept recumbent, and then sent to a stage II PACU where he is encouraged to sit up and ambulate. When patients are awake, oriented, and able to walk, they may have liquids offered to them by mouth, and be encouraged to ambulate to the bathroom and attempt to void. If this is all easily accomplished and patients have no excessive pain, nausea, or vomiting, they are considered

Table 6. *Stages of Recovery and Some Tests for Their Assessment*

STAGE OF RECOVERY	TEST OF RECOVERY
Awakening and recovery of vital reflexes	Patient can open eyes and answer questions and can maintain and guard his or her own airway
Immediate clinical recovery	Patient can sit and stand unaided
Home readiness	Patient can walk on a straight line, paper and pencil tests, and simple coordination and reaction tests
Street fitness	Psychomotor test batteries
Full recovery (complete recovery)	Carefully selected psychomotor test batteries and real driving tests
Psychological recovery	Psychological tests

From Kortilla K: Practical discharge criteria. *In* Wetchler BV (ed): Problems in Anesthesia: Outpatient Anesthesia. Philadelphia, Lippincott, 1988, pp 114–151; with permission.

"home ready" and may be discharged with an escort. The escorting person should remain with the patient overnight. The need to demonstrate the ability to void is not a strict requirement for patients who have undergone a general anesthetic, but patients who have had spinal or epidural anesthesia should demonstrate that they can void before they are allowed to go home (Table 7).

Prior to discharge, patients should have their dressing checked,

Table 7. *Guidelines for Safe Discharge after Day Surgery*

1. Patient must have a responsible "vested" adult to escort patient home and provide care at home
2. Patient's vital signs must have been stable for at least 1 hour
3. Patient must have no evidence of respiratory depression
4. Patient must be
    Oriented to person, place, time
    Able to dress himself/herself
    Able to walk out without assistance
    Able to maintain orally administered fluids*
    Able to void*
5. Patient must have
    More than minimal nausea or vomiting
    Excessive pain
    Bleeding
6. Patient should stay at least 1 to 2 hours after extubation*
7. Patient must be discharged by the person who gave the anesthesia or a designee. Written instructions for the postoperative period at home including a contact place and person need to be reinforced

* The role of these variables as criteria for discharge remains to be established.

From Kortilla K: Practical discharge criteria. *In* Wetchler BV (ed): Problems in Anesthesia: Outpatient Anesthesia. Philadelphia, Lippincott, 1988, pp 144–151; with permission.

and written instructions should be given in the presence of the responsible person. They must be reminded not to drive, operate power tools, or be involved in major decisions for up to 24 hours after their anesthetic.

Patients should also be informed about the common side effects postanesthesia and postsurgery that might be expected, in order to allay anxiety associated with these symptoms. Patients should be told to expect mild pain, nausea and vomiting, sore throat, muscle aches, and headache. The patients should be given the telephone numbers of the surgeon, the ambulatory center, and the emergency room, should problems arise after they are discharged.

New and revised standards by the Joint Commission on Accreditation of Health Care Organizations in the 1988 edition of the Accreditation Manual for Hospitals do not require a physician to examine the patient just prior to discharge. The 1988 standards require a licensed independent practitioner to have relevant discharge criteria rigorously applied in order to determine the readiness of the patient for discharge. When the patient meets these criteria, the patient can be discharged by the staff. An example of these criteria is listed in Table 8.

Table 8. *Checklist of Discharge Criteria at Rush Ambulatory Surgery Unit*

DISCHARGE SUMMARY	
Progress Notes:	Discharge Criteria
	___ Voided (if applicable)
	___ Patient given general written post-op instructions
	___ Alert and oriented
	___ Vital signs stable
	___ No excess bleeding or drainage
	___ Minimal nausea and vomiting
	___ Able to ambulate (except ortho cases)
	___ No pain medications in ½ hour
	___ Discharged to a responsible adult
Specific discharge instructions:	___ Minimal postoperative pain
	___ Ambulatory Recovery Post Anesthesia score totals 10 (if applicable)
Discharge vital signs: B/P T P R	Discharge Time ___
Postoperative phone call desired: Yes ☐ no ☐	Signature M.D. Surgeon
I hereby accept, understand and can verbalize the discharge instructions.	Signature M.D. Anesthesiologist
Patient/Parent	Signature R.N.

Table 9. *Postdischarge Follow-up Form at Rush Ambulatory Surgery Unit*

FOLLOW-UP CONTACT:

	Yes	No	N/A	Comments
Hoarseness/Cough				
Elevated temp.				
Nausea/Vomiting				
Bleeding				
Dizziness				
Pain				
Drainage				

Other: ______________________________

Date ________ Time ________ 1st call ______________________ R.N.
Date ________ Time ________ If unable to contact on first attempt,
2nd call ______________________ R.N.

Finally, facilities should have a method of follow-up for their patients after discharge. Telephone calls the day following surgery or postcard evaluations are the most common methods used. These follow-ups help centers get feedback on their performances as well as allow patients the opportunity to express their concerns regarding their ambulatory surgery experience (Table 9). It must be remembered, above all else, that when it is unclear whether a patient is ready for discharge, a physician should examine the patient, as clinical judgment is the most important indicator of readiness for discharge.

SUMMARY

We have discussed the problems that are seen in an ambulatory surgery practice, and the different anesthetic techniques that can help to avoid specific problems. The two main problems in the PACU are the control of pain and the control of nausea and vomiting.

The control of pain postoperatively in the ambulatory surgery setting can be accomplished in a number of different ways. Premedication or supplementation of anesthesia with potent intravenous opioid analgesics can significantly decrease postoperative pain and enhance recovery, as well as speed up patient discharge times. The smallest doses that are effective should be employed to minimize problems such as nausea, vomiting, and drowsiness. When possible, infiltration with local anesthetics or regional blocks can be employed either alone or in combination with potent intravenous narcotics to allow the greatest patient comfort in the postoperative setting, with the least delay in discharge.

Postoperative nausea and vomiting remains an important and clinically significant problem for anesthesiologists, the PACU nurse, and patients. Management of the problem needs, first and foremost, to be centered around prevention, placing the primary responsibility on the anesthesia team. Prompt treatment in the PACU requires a cooperative effort among the anesthesiologist, the PACU nurse, and the patient, all geared toward the goal of decreasing or preventing morbidity and improving patient safety, comfort, and recovery. We now have agents that are effective in both prophylaxis and treatment of postoperative nausea and vomiting, as well as an increasing knowledge of which anesthetic techniques need to be avoided. When supplemented with rapid pain control in the PACU, and good communication between the ambulatory surgical staff and the patients and their families, we can minimize the disruption that postoperative nausea and vomiting can create in the ambulatory surgery experience.

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Legal Problems in the Postanesthesia Care Unit

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> Justice, justice shalt thou pursue.
>
> DEUTERONOMY 16:20

The anesthesiologist's practice has been immeasurably eased by development of the recovery room.[14] Here, postanesthesia issues involving the return of sensorium, pain, and fluid-electrolyte balance must be addressed. Often, problems originating in the operating room become manifest (blood loss, neuromuscular blockade, and so forth) and must be treated. Here, also, we see specific medicolegal risk management issues. Although it is not the objective of this presentation to review "the law" exhaustively, we will focus on some of the most pertinent administrative and legal situations facing the recovery room director.[12, 15]

At this point it is also pertinent to note that the cited lower court cases are of a descriptive value only. They should not be mistaken as establishing precedents (stare decisis) since they have not been reviewed by higher, appellate, or supreme courts. In fact, in most jurisdictions "published" accounts are not even available, with the exception of local "unofficial" reporters. Copies of the court records by docket numbers and the date of decision can usually be obtained only by referring to these "unofficial" reporters or on application to the clerk of the specific court.

To state the four elements for negligence actions at law is necessary. First, there must have been a duty, a responsibility to act. Second, there must have been a breach or failure of that duty, i.e., a

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violation of the "standard of care." Third, this breach must have been the proximate cause of plaintiff's injury. Last, the plaintiff must show actual harm that must have been foreseeable or reasonably anticipated in a related chain of events.

It is equally important for the physician reader of this discussion to recognize that in negligence cases the issue of intent to do harm or do good is not a factor. Similarly, intent and motive are not to be confused. Intent refers only to the state of mind with which the act is achieved or outlined. Motive is what prompts a person to act or fail to act.

AUTHORITY

A complicated legal problem is the authority under which a physician can delegate care to a nonphysician. In most jurisdictions anesthesiology is considered to be the practice of medicine and thus can be practiced by a nonphysician only under delegation. For example, in Michigan the delegatory authority is defined in the Public Health Code (Public Act 368 of 1978, as amended). "Delegation" is defined as

> . . . an authorization granted by a licensee to a licensed or unlicensed individual to perform selected acts, tasks, or functions which fall within the scope of practice of the delegator and which are not within the scope of practice of the delegatee and which, in the absence of the authorization, would constitute illegal practice of a licensed profession.[13]

Section 333.16215 of the Michigan Compiled Laws states the following:

> (1) A licensee . . . may delegate to a licensed or unlicensed individual who is otherwise qualified by education, training or experience the performance of selected acts, tasks, or functions where the acts, tasks, or functions fall within the scope of practice of the licensee's profession and will be performed under the licensee's supervising. An act, task, or function shall not be delegated under this section which, under standards of acceptable and prevailing practice, requires the level of education, skill and judgement required of a licensee under this article.

Note that the clause is clearly limited by not permitting delegation if the *acceptable* standards of practice require a level of education, skill, and judgement that the delegator must have. What is inherent to this reasoning is that the delegator (attending physician) has a clear duty to supervise the delegatee (house officer, nurse). This becomes even more crucial when we address Section 333.16221, referring to the Department of Licensure and Regulation's authority to investigate (and inherently to discipline) the licensee's practice of the health profession. Subsection (a) includes the following:

> A violation of general duty, consisting of negligence or failure to exercise due care, including negligent delegation to or supervision of employees or other individuals, *whether or not injury results*, or any conduct, practice, or condition which impairs, or may impair, the ability to safely and skillfully practice the health profession.

We see here that the standard of care and evidentiary standards in an administrative medical board disciplinary hearing are less constrained than in a medical malpractice trial. The law may vary somewhat from state to state. The prudent practitioner will become conversant with the provisions of statutes governing the jurisdiction wherein he or she practices.

There is some question as to who the delegator may be in an anesthesia situation. If there is no anesthesiologist on the hospital staff (a situation that regrettably is not at all uncommon), the attending surgeon is liable for any anesthesia misadventure. The determination of where the liability lies in a disputed situation is for the finder of fact, i.e., the jury, to determine.

In the postanesthesia care unit (PACU), leaving the patient in the care of the nurse may or may not be a question of delegation, depending on the circumstances, the actions the nurse is asked to perform, the procedure and policy document of the PACU, the training of the nurse, and a number of other variables.

In general, the anesthesia person responsible for the management of the anesthesia is responsible in the postoperative period, at least during the time that the patient remains in the PACU, unless the patient has been turned over to and accepted by another person of comparable skill and experience. We do not believe that a PACU nurse can accept the responsibility for the patient as an independent health care practitioner other than under the delegatory clause of the code. Thus, the ultimate liability will remain with the anesthesiologist or the surgeon, notwithstanding hospital liability for the certified nurse anesthetist (CRNA) employee. Once the patient is discharged from the PACU, the liability reverts from the anesthesiologist to the surgeon, except for directly related anesthesia problems, which remain under the purview of the anesthesiologist or, at the very least, become a shared responsibility with the surgeon.

PATIENT MANAGEMENT

The basis of most management problems in the postanesthesia unit rests in close observation of the patient. Thus, we address monitoring procedures and staffing of the unit. The anesthesiologist is most likely to be subject to legal liability in connection with postoperative care because of (1) failure to monitor a patient's recovery from the anesthetic procedure adequately, (2) the use of an inappropriate drug or procedure during the recovery period, or (3) premature discharge of a patient from the PACU. Similarly, the question of who is in charge, founded on practices of transfer of patient care, is of crucial import.

The anesthesiologist's control of immediate patient physiologic management as a member of the operating room team is no longer an issue. However, when the patient arrives in the PACU, the nursing staff takes over for continuing care, presumably under medical supervision.[13] Sufficiency of staff involves questions of training, patient-nurse ratio, and monitoring. Standards generally discussed suggest the following: "One professional registered nurse will be responsible for not more than two patients. . . . Depending on the level of care needed, a higher ratio may be necessary."

We must remember that postanesthesia care, as defined by the American Society of Anesthesiologists (ASA), is multifold, and includes, but is not limited to, the obligation for a member of the anesthesia care team to accompany the patient to the PACU, document patient status on arrival, transmit information concerning the preoperative condition and the surgicoanesthetic course, and remain with the patient until the nurse accepts nursing care responsibility. Obviously the latter includes a mandate to remain until the patient's acute needs are met as well. Previous guidelines[3] were phrased according to the following outline. Postanesthesia care means the following:

1. Availability of adequate nursing personnel and pertinent equipment necessary for safe postanesthesia care.
2. Awareness by responsible physicians of competence level of personnel who carry out postanesthesia care.
3. Informing personnel caring for patients in immediate postanesthetic period of any specific problems presented by each patient.
4. The individual responsible for administering anesthesia remains with the patient as long as his or her presence appears necessary.
5. A physician participates in establishing policies for the discharge of patients from any postanesthesia facility.
6. The physician should review the patient's status on at least one visit, with appropriate notation on the patient's chart during the early postanesthesia period if feasible.
7. Provision of management of related anesthetic complications.

Along with these guidelines were the following[22]: (1) evaluation of the status of the patient on admission and discharge from the PACU; (2) a record of vital signs; (3) a record of intravenous fluids administered, including blood and blood products; (4) a record of all drugs employed; (5) a record of unusual events or postoperative complications including management thereof; and (6) the anesthesiologist shall determine and document when the period of postoperative surveillance has terminated.

It is interesting to note that the above formulations of guidelines were subsequently modified to five standards.[21] The five standards are presented:

Standard I. All patients who have received general anesthesia, regional anesthesia, or monitored anesthesia care shall receive appropriate postanesthesia management.

Standard II. A patient transported to the PACU shall be accompanied by a member of the anesthesia care team who is knowledgeable about the patient's condition. The patient shall be continually evaluated and treated during transport with monitoring and support appropriate to the patient's condition.

Standard III. Upon arrival in the PACU the patient shall be reevaluated and a verbal report provided to the responsible PACU nurse by the member of the anesthesia care team who accompanies the patient.

Standard IV. The patient's condition shall be evaluated continually in the PACU.

Standard V. A physician is responsible for the discharge of the patient from the postanesthesia care unit.

The authors leave it to the reader to note the differences in emphasis, and perhaps intent, for implied shifts in liability exposure. The ASA is noted to be a voluntary organization and the above formulations are presented as guidelines or recommendations and not as law, yet the listing is noted as "standards." The courts recognize (by taking "judicial notice") national professional groups as quasi-judicial bodies wherein the profession purportedly is regulating itself (i.e., its members) for the benefit of its patients. As such the "recommendations" have the evidentiary impact of law. On the other hand, modification of previously detailed recommendations may be looked on as self-serving "waffling" and should not be considered by the practitioner as lessening of the earlier, more specific guidance. In fact, the new wording may expand liability by the very vagueness or expansiveness of the phraseology.

A new pattern is arising that is beginning to affect hospitals and that ultimately will affect every aspect of high-intensity care practice. This is the growing development and explicit delineation of "standards" by insurance companies and government. At the present the standards relate to "patient dumping," an important aspect of interinstitutional patient transfer. However, the standards also establish a federal standard of care for medical malpractice,[8] and create a strict liability cause of action against hospitals that violate the standards. The most interesting consideration is that, for the first time, a federal standard of care for medical malpractice is established. As elucidated in a recent case[18] it emphasized the need for adequate space for the patient and staff on hand who were qualified to handle the problem. As an indication of the trends in health law we must keep in mind that anyone who suffers "personal harm" as a result of violation of the statute—including, presumably, relatives of the patient—can collect "those damages available for personal injury" under state law. Therefore, "negligence" is becoming irrelevant; hospitals are strictly liable whenever there is a statutory violation. This also does away with the defense argument that the physician is "independent," making the hospital liable even if the doctor was in fact an independent contractor in control of his own judgements and actions. Note the similarity to the *Falcon* v. *Kelso* case (see following), which is another step toward stricter liability concepts in medical malpractice.

Ancillary staff must include administration, engineering, electrocardiography, laboratory, pharmacy, physical therapy, radiology, respiratory therapy, and social services. All members of this team rely on monitoring of varying technologic intensity. Electrocardiograms, multichannel arterial, venous, and/or pulmonary arterial pressures, recording pulse oximetry, as well as intracranial pressure measurements are standard of care. Blood gas analyses are concomitant features. Many monitors have alarms capable of detecting obstruction, disconnect, arrhythmia, and other features.

Practitioners consider all monitors as ancillary aids to their medical judgement. Attorneys view this information as providing the earliest notice that something may be wrong. In legal parlance, this may be the "last clear chance" to be alerted to problems requiring protective action. Further, hospital corporate responsibilities have expanded to the extent that liability exists if a hospital does not have the equipment, facilities, and personnel necessary for the care of a patient.[6] A recent Michigan Court of Appeals decision *Falcon* v. *Kelso*[9] (on appeal) represents a herald sign of alternatives to the last clear chance doctrine. This refers to the concept that the monitoring, intravenous lines, et cetera was the last chance for being alerted to impending disaster. In this case the court (which does not formally recognize the last chance doctrine) expanded the concept of "proximate cause." In *Falcon* the plaintiff alleged that the physician's failure to have an intravenous line in place *reduced the patient's chances* of survival. The court ruled that plaintiffs in a medical malpractice case need only show that a patient would have had a *better chance* of survival absent a physician's omission. The court thus rejected the standard of that jurisdiction that required plaintiffs to prove *probability* of survival, making it much easier to prove proximate cause in medical malpractice cases.

The import of this discussion relates to the diverse meaning of "probable" and "proximate" causation. First, the phrase "probable cause" is often misused as synonymous with "proximate causation." Probable cause (usually used in criminal law parlance) strictly should be understood in a civil case as a cause of action existing based on an apparent state of facts found to exist upon reasonable inquiry. Proximate causation, as is more frequently applied in a negligence case, is that which stands next in causation to the effect, not necessarily in time or space but which in a natural and continuous sequence produces the injury that might be reasonably anticipated. The interpretive expansion of the Michigan court thus represents a further step toward imposition of strict liability. If this is indeed a portent of the future, the only real issue at trial may be the amount of damages to award plaintiffs!

WHO IS IN CHARGE?

Another area of medical contention is "who is in charge?" We are often misled by an administrative structure specifying that the PACU is to be under the direction of the anesthesiologist, surgeon, or intensivist. It is apparent that no one physician is competent to manage all aspects of every patient's care. Still we must recognize that the anesthesiologist has primarily addressed pathophysiologic, pharmacologic, and other patient needs in the operating theater.

In transferring the patient to the PACU (thus freeing the anesthesiologist to go on to another patient's care) certain principles must be met. First, the physician must not leave the patient as long as acute needs dictate the necessity for professional attendance. To leave may represent an abandonment of the patient. In *Ricks* v. *Budge*[19] the court held that a physician, upon undertaking a procedure, is under the duty of continuing attention as long as the case requires such attention. Provision of adequate alternative medical coverage is permissible but requires fulfilling certain criteria for transfer of the patient. This is of importance because (1) the attending physician may have to leave the hospital before the patient is discharged from the PACU; (2) postoperative care may involve overlapping responsibilities and liabilities; (3) disparities in training, experience, and professional status may require consideration; (4) the patient usually contracts for care with a specific physician (e.g., surgeon), but there is an implied contract with anesthesia personnel and with the hospital for ancillary care professionals; and (5) patients in the PACU are inherently unstable.

The basic issues are transference of patient care to an "equally qualified" professional and availability for necessities of care during this crucial phase.[1] The person to whom the patient is transferred must not only be immediately available but must accept the transfer. He or she must be equally competent. Note that this transfer assumes that the patient does not have an immediate critical need. *Bronson* v. *Sisters of Mercy* involved just such a situation.[5] The patient was a middle-aged woman who was scheduled for a series of epidural steroid administrations. The anesthesiologist who did the epidural with a mixture of hydrocortisone and a long-acting local anesthetic took the patient to the PACU, wheeled her through the door, told the nurse to attend to the patient, *and left.* When the nurse tried to take the vital signs, she found that the patient was not breathing and had no measurable blood pressure. Resuscitation was started promptly but the patient was decerebrate and died several weeks later. In this case there was an improper transfer, an abandonment.

Another aspect of assumption of care by the PACU staff is illustrated by *Beauchamp* v. *Sparrow Hospital.*[4] Beauchamp was a 20-year-old woman who suffered a fractured kidney in a motor vehicle accident. She had a nephrectomy and was taken to the PACU in good condition. At that time, the policy of the hospital unit was that no nurse was responsible for any specific patient and that all patients were under the care of all nurses. On that particular day, matters were further complicated by a going-away party for one of the nurses so that, in rotation, one or more nurses left the PACU. When a nurse finally went to check on Beauchamp the patient was dead. The medical examiner found that death was due to airway obstruction. The hospital was found liable for very heavy damages. Implicit in this case are the administrative questions as to overall supervision of the unit, the number of patients per nurse, specific mechanisms of transfer of care of patients entering the PACU from the operating room, the ratio of nurse to patients, and other issues.

Nurses have a primary duty to protect the patient. This implies adequate staffing. A hospital's failure to provide adequate assistance in the immediate postoperative period was discussed in the Czubinski case[7] in which the court stated that even leaving one patient to serve another or for other required duties cannot be permitted and constitutes abandonment.

Another classic example is that of the Goldsby case.[10] Goldsby was a young man who was mugged and had his mandible broken. He was admitted to the hospital and scheduled for an open reduction and internal fixation of the mandibular fracture. This was performed under general endotracheal anesthesia. At the end of the procedure the jaws were wired shut. The patient was extubated in the operating room. He was breathing well and was responding to stimulation. The patient was taken to the PACU and the nurse was informed about the wired jaw. Postoperative orders clearly stated that the patient should be in a semi-sitting position and that wire cutters must be at the bedside. The anesthesiologist left to do another case. As soon as he left the PACU the nurses also left for an in-service lecture, leaving an operating room nurse in charge of the patients. When she made her rounds 10 minutes later, she found Goldsby without pulse or blood pressure. The cardiac arrest code was called but the patient could not be resuscitated. Goldsby was flat in bed and there was no wire cutter in the PACU. The hospital was found negligent, but the anesthesiologist was not. This tragic accumulation of errors highlights problems of communication of information, failure to follow orders, patient abandonment (improper transfer of care), and administrative staffing error. The staffing issue relates to provision of adequate help while regularly assigned personnel are required to be out of the unit for in-service lectures. The transfer of care concept requiring "equally qualified" expertise is just as applicable for nurses as for physicians. Protection of the patient is sine qua non. Certainly, transfer of orders applies from nurse to nurse and is a standard of care of nursing practice. How frustrating to await for a specific nurse or chart during a change of shift! Is it not the responsibility of administrators, lay or physician, to take precautions and establish proper rules and protocol with patient protection as the object?

Of no lesser import is the communication of the PACU nurse with the floor or room nurse when the patient is transferred back to his or her room. This relaying of vital data, orders, reports of adverse reactions and other complications, et cetera, resulting in proper continuation of care, is crucial in a period of high vulnerability. And, it *must be documented, including a formal acceptance of the transfer.*

Triage usually refers to evaluating and sending patients to a care unit; however, a patient can be transferred out of that unit. Often discharge from the PACU receives little attention. We have made great strides in recognizing that a physician, rather than a nurse, discharges the patient from the PACU. Transfer to "floor care" is another matter not from the discharge aspect, but regarding those individuals receiving the transfer. The same requirements apply as for any other transfer. The problem often seen is the inability to locate the proper nurse to report to and one who is willing and competent to accept the patient. It is important to affirmatively and specifically inquire of the nurse "do you understand this patient's problems and do you accept this patient's transfer to your responsibility?" This is to re-emphasize the special legal, ethical, and moral considerations that pertain when transferring surgical patients to nonsurgical care units for whatever reason.

TRIAGE

Since the advent of the PACU concept,[11] we have always had the problem of space allocation. Too frequently there is a back-up in this area often attributed to keeping the patients in the unit too long or improper utilization of the allocated spaces. With the advent of Diagnosis-Related Groups, forced on the hospitals and the medical profession by government, renewed consideration must be given to triage. This has come about because institutions have been constrained to seek ways to decrease costs. At times the result threatens quality of care. In spite of this, PACU space and equipment facilities may be limited at one end of the scale, while pressure to increase operating room utilization increases.

At the other end of the spectrum, there is implicit pressure to get patients out of the PACU "as soon as possible" (translated: pressure to release patients, perhaps before they are ready to leave). We will not discuss ethical and moral factors that the courts have skirted to date. A first-come first-serve approach for PACU admission would appear to be indicated. This process does not address cost-effective relationships. It also does not recognize current trends in hospital care, namely that floor care is essentially custodial. Perhaps we need to revert to concepts of special duty nursing or further develop intermediate care units. The preferred approach is for the patient to receive that level of attention that is responsive to the needs of the patient and excludes the administrative problems presented.

We must emphasize that to depart from customary postoperative procedures, when circumstances require, does not necessarily constitute negligence. The case of Prack[17] illustrates this point. The patient was returned to her room on the completion of surgery rather than to the PACU. The PACU was closed for the evening because of staffing considerations. The patient's room was provided with a qualified nurse employed specifically to care for this postoperative patient. After accompanying the patient to her room, the anesthesiologist reintubated her, attached the Levin tube to a suction device, affixed an oxygen nasal catheter, and, after assuring himself that the patient's condition was satisfactory, left to return to the operating suite for another case. Soon afterward the patient suffered a cardiac arrest, and the anesthesiologist was requested to bring a ventilator from the operating room. He was delayed several minutes by his duties with the second patient. Prack was cardioresuscitated but never regained consciousness.

At trial, the verdict favored the physician-defendant and the hospital. The appellate court, upholding the hospital's failure to provide a room for postanesthesia care, found that (1) hospitals in that area customarily closed their PACU at night; (2) the anesthesiologist would not have been available in the PACU either, due to his duties with the second patient; and (3) the anesthesiologist could not reasonably expect that the prior patient would need his continued attention.

Although this case does support our basic point, serious doubts remain about parts of the decision. First, why was it necessary to reintubate a patient immediately after transfer to her room? Next, what was achieved by affixing an oxygen nasal catheter to a patient with an endotracheal tube in situ (why not oxygen via the endotracheal tube)? In 1990, the "area practice" argument for hospital facilities would also have lesser weight.

Another quoted case demonstrates the more prevalent approach, that extenuating circumstances would be required to justify failure to provide full postoperative care including ancillary personnel, equipment, and professional supervision. The Pederson case[20] involved a dentist-surgeon assisted by a nurse anesthetist. Hospital regulations required a physician to admit the patient and to be responsible for the management of any medical condition of the patient. No physician attended the operation, and no other physicians were present in this small hospital. After surgery, and while the patient was in the PACU, the patient suffered convulsive seizures. There was an extensive delay before the physician responded. The patient suffered disabling brain damage. The court stated that there was negligence as a matter of law in permitting the operation to take place without the immediate availability of an attending physician. The court strongly relied on the hospital's own rules and regulations. Perhaps the most important lesson in this case is the absolute obligation to follow rules and regulations promulgated by the hospital because they become the "standard of care" for the institution.

In conclusion, we have presented some of the most cogent medicolegal problems of the PACU. This is not all inclusive. For that we refer the reader to previously mentioned reference sources. We hope that this discussion will stimulate further interest and study.

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